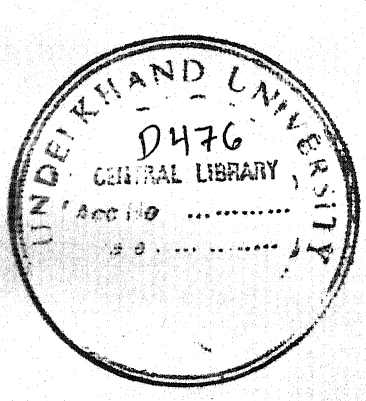
"TO FIND OUT THE DAY TO DAY VARIATION IN BASAL CHOLESTEROL AND OTHER LIPOPROTIENS IN INDIAN POPULATION"

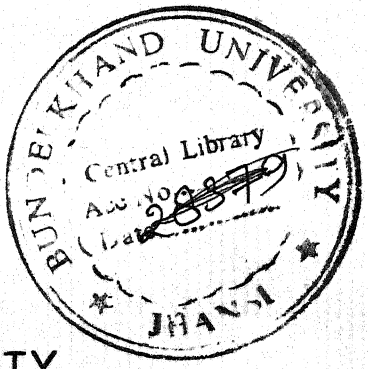
THESIS

FOR

DOCTOR OF MEDICINE

(MEDICINE)





BUNDELKHAND UNIVERSITY
JHANSI (U. P.)

1996

RAM SEWAK

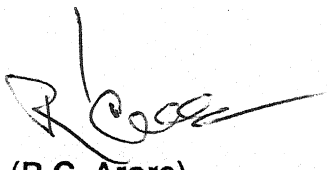
Dedicated
to
my parents
&
my most
respected & affectionate
teachers

CERTIFICATE

This is to certify that the work entitled"**TO FIND OUT THE DAY TO DAY VARIATION IN BASAL CHOLESTEROL IN INDIAN POPULATION**" which is being submitted as a thesis for M.D. (Medicine) Examination, 1996 of Bundelkhand University by **Dr. RAM SEWAK** has been carried out in the department of medicine, M.L.B. Medical College, Jhansi.

He has put in the necessary stay in the department as per university regulations.

Dated :



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CERTIFICATE

This is to certify that the work entitled"**TO FIND OUT THE DAY TO DAY VARIATION IN BASAL CHOLESTEROL AND OTHER LIPOPROTEIN IN INDIAN HEALTHY POPULATION**" which is being submitted as a thesis for M.D. (Medicine) Examination, 1996 of Bundelkhand University, has been carried out by **Dr. RAM SEWAK** under my direct supervision and guidance. The techniques embodied in the thesis were undertakan by the candidated himself and observations recorded have been checked by me from time to time.

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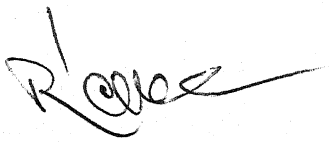

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Date 30/11/95

(Dr. Ram Sewak)

CONTENTS

INTRODUCTION

INTRODUCTION

The Hyperlipoproteinemia are disterbances of lipid transport that result from accelerated synthesis or retared degradation of lipoproteins that transport cholesterol and triglycerid through Plasma.

Cholesterol is a complex Monohydric secondary alcohol stable white crystalline substance, insoluble in water but readily soluble in non polar solvents, chemically basic structure consist of 27 carbon atom ring referred to as cyclopenteno Perhydrophenanthrine ring two-third of cholesterol in blood is found in esterified form while 1/3 rd is present as free cholesterol.

Plasma lipid is mixture of cholesterol, triglycerides, phospholipid and protein that remain in direct contact with intima any change in them would be correlated best with disease (Rritchevsky, 1964). Effect of dietary cholesterol has not been considered significant in man according to page et al (1965) stated that " Low fat diet has no role in the treatment of myocadial infarction.

In 1982, a quantitative analysis of epicardial coronary arteries was carried out by Heney Scott et al. It showed that left main coronary artery was narrowed more significantly in subjects with hypercholesterolemia or hypertriglyceridemia, than in those with normal STC and STG levels. A significant relation was found between STG and amount of severe coronary narrowing but none between STC and amount of severe narrowing.

Laren (1966) observed that a recduction in dietary cholesterol however has no change in subjects above 60 years of age. When po;yunsaturated fat have been substituted for saturated fats in diet, it will lower serum cholesterol but not the serum trighlyceride (Ahrens et al, 1957 ; Kinsell et al, 1952 ; Grundy, 1995).

The problem of transporting cholesterol and triglycerids through the aqueus environment of plasma was solved by enveloping these hydrophobic neutral lipids in a coat of phospholipid and special proteins (called apolipoproteins) with the characteristic properties of detergents. The phospholipid molecules are arranged on surface of lipoproteins with their polar head groups facing the surrounding aqueous environment and there fatty acid side chains penitrating the interior of particles.

One Exogenous Pathway is responsible for digestion, absorption and tissue disemination of dietary fat, the lipoproteins are globular particles of high molecular weight that transport non polar lipids (Primarily triglycerides and cholestrol esters) through plasma.

A reduction in cholesterol carrying lipoprotein achieved by diet & drugs, reduces the risk of myocardial infarction in subjects with Hyperlipoproteinemia. Largest amounts of lipoprotein are invloved in transport of dietary fats which amount to be more than 100gm triglycerid & about 1 gm cholesterol/day within intestinal epithilial cells dietary triglycerides and cholesterol are incorporated into large lipoprotein particles called chylomicron.

The chylomicrons are secreted into intestinal lymph and passes into general circulation for transport to capillaries of adipose and skeltal muscles where they adhere the binding sites on capillary walls.

The liver also distributes cholesterol to other tissue *BY ENDOGENOUS PATHWAY*, very low density lipoprotein **(VLDL)** particles are relatively large carry 5 to 10 times more triglycerides than cholesterol esters and contain a form of apoprotein B designated B100 that differs from apoprotein B 48 of chylomicrons.

About 3/4th of total cholesterol in normal human palsma is contained within LDL particles.

In human 70% to 80% of LDL is removed from palsma by LDL receptor pathway.

Plasma cholesterol and triglycerides levels provide information regarding the nature of lipoprotien particles that is increased, on isolated elevation in plasma triglycerides indicates the concentration of chylomicrons or VLDL are increased, on other hand, an isolated elevation of palsma cholesterol nearly also indicates that the concentration of LDL is also increased.

As a working rule hyperlipoproteinemia is considered to be present whenever the palsma cholesterol level exceeds 200 mg/dl or triglycerides levels exceeds 200 mg/dl.

When total cholesterol level exceeds 240 mg/dl, HDL levels should be measured since low level of this lipoprotein class are statistically associated with increased risk of myocardial infarction.

Classification of total cholesterol and LDL cholesterol values.

	Total plasma cholesterol	LDL cholesterol
Desirable	----- < 200 mg/dl	------ < 130 mg/dl
Border line high	----- 200 - 239 / dl	------ 130 - 359 mg/dl
High	----- > 240 mg/dl	------ > 160 mg/dl

[Report of national cholesterol education program expert panel on detection evaluation and treatment of high blood cholesterol in adults. Arch Intern Medicine 148 : 36 ; 1988]

It has been seen that hypercholesterolemia is associated with atherosclerosis and it contributes certain diseases like ischemic heart disease, cerebrovascular accident (CVA) and acute pancreatitis, as working rules high level of cholesterol to be present when plasma level exceeds 200 mg/dl. High level of high density lipoproteins (HDL) is associated with decrease the risk of myocardial infraction.

It has been seen that serum cholesterol is variable with certain varies with age, sex, genetic factor, smoking, emotional stress, physical activity hypertension, obesity, diabetic mellitus and certain drugs like estrogens, oral C pills, glueocorticocoids and alcohal intake etc. It is difficult to yield some absolute values of same subject by using different methods to estimate serum cholesterol the Biological & technical variability of serum lipids has been reported as early as 1954 by watkin et. al.

There is hardly any consensus among physicians about the level of cholesterol which is safe and after which particular level intervention

is needed. It is common practice in U.S. for universal screening for cholesterol count but even in countries like U.K. universal screening is not practice.

Recent studies have proved that on repected testing 40% of individuals can shift from high risk zone to safe zone & vice versa. This has also been observed by Thompson et. al (1990).

Classification of Hyperlipoproteinemia

This classification attributed to Fredrickson and his collegues is still clinically valuable but fail to identify the molecular or genetic defect(s) responsible for condition. Consequently patients with the same genetic defect may fall into two or mor of five Fredrickson categories ; and progression of the condition or treatment may cause a patient to move from one category to another.

Familiar hypercholesterolemia is best defined clinically, genetically and biochemically disorder characterized by (a) deposition of LDL derived cholesterol in normal sites in the body (c) inheritance as an autosomal dominant trait. It occurs at a frequency of about 1 in 100 person.

Classification of the Hyperlipoproteinemia

Fridrickson Type	Genetic Classification	Defect	Risk
I.	Lipoprotein lipase deficiency	Mutated or absent lipo-protein lipase	Pancreatitis
	APO CII deficiency	Mutated lipoprotein lipase cofactor	
II.	Familial hypercholesterolemia	Diminished or absent	CHD
	APO B3500 defect	LDL receptor interaction	
III.	APO E_2 homozygocity	Mutated apolipoprotein E plus precipitating environ mental factor	CHD and PVD
IV.	Familial combined Hyperlipidemia	? ----------	CHD
	Familial Hypertriglyceridaemia	? ----------	Pancreatitis
V.	Familial Hypertriglyceridaemia	? ----------	Pancreatitis

CHD = Coronary heart disease, PVD = Peripheral vascular disease.

Recently attampts have been made to introduce a genetic classification system but as different molecular defects are being discovered, this approach has become increasingly complex. So until gene therpy or pharmacologic manipulation of molecular defects become real possibilities, genetic classification are unlikely to prove widely useful in clinical practice & most Physicians will remain loyal to the older Fredrickson classification.

Present study has been proposed to find out the day to day variation in basal lipoprotein in healthy subjects.

REVIEW OF LITERATURE

Review of Literature

Recent studies suggest that low density lipoprotein (LDL) are main culprit of otherosclerosis. LDL are rapidily taken up by arterial smooth muscles and arterial endothelial cells [Rose et. al 1976]. Serum low density lipoprotein (LDL) are main carrier of cholesterol and apoproteins both which have been found in Atherosclerotic Patch [Hoff et. al 1973].

Plasma lipid and lipoprotein concentration in man and woman vary with age, total cholesterol value are higher in man as compared to woman between age 20-55 year. Values of VLDL are similar in both sex at 20-59 years, Values of HDL are higher in woman then man through out the age range.

High density lipoprotein HDL is protective in nature, major role of HDL is centripetal transport of cholesterol from peripheral tissue to liver (clornset, 1963) and indirectly it facilitates transport of triglycerids.

Woman taking sex harmones have higher total cholesterol then nontakers between age 20-50 years. In chronic renal diseases the serum cholesterol and serum triglyceride tend to be elevated. Elevation is inversely related with serum albumin level more than 40% of young patients of documented coronary artery disease are normal cholesterolemic (Gregory et. al. 1983).

Chiba et al (1984) observed variability of serum high density lipoprotein cholesterol concentration in healthy subjects in 3 years term

and find that mean HDL and serum total cholesterol values varied to some extent year by year when effect of age and menopause was eliminated they concluded that non mormolipidemic people had winder individual variation both in high density Lipoprotein (HDL) and triglyceride.

Natelson and others: Concentration of Serum cholesterol can vary during day both serum cholesterol (STC) & High density lipoprotein (HDL) cholesterol concentration to be in evening and lowest in early hours of morning shows diurnal variation.

Heggsted DM et al (1987). observed a large difference in serum cholesterol level of same subject by unknown reason. In a single sample from a man with mean serum cholesterol level 220 mg/dl can be expected to fall between 200 and 220 mg/dl - A RANGE FROM NO RISK TO HIGH RISK. Many individual may show greater variation then this [Blank et al 1986] variety of methods are available to estimate serum cholesterol but may not yield same absolute values.

In the studies of our department [Arora & Sharma 1984]. The effect of high cholesterol fat diet after single dose was studied in healthy subjects & subjects of ischemic heart disease, diabetes mellitus and chronic renal disease. Post prandial changes were observed in serum total cholesterol, low density lipoprotein and serum triglycerides.

[Rose and Glomet 1976] : It has been found that in atherosclerotic lesion there is smooth muscle proliferation along with large amount of connective tissue matrix and intracellular and extracellular lipid.

Each cell responds to different atherosclerosis lies in the intima of vessels in form of fatty streaks of Plaque [Gill 1977].

In a study carried out in our department Arora & Umashanker (1993) majority of healthy effects of single high cholesterol fat diet was observed on healthy individuals and patients of diabetic mellitis Hypertension and ischemic heart disease. Majority of healthy population showed a fall in serum total cholesterol and low density lipoprotein(LDL) at one hour & patients of diabetes mellitus, Hypertension & ischemic heart disease minority of healthy subjects revealed rise in serum total cholesterol (STC) and low density lipoprotien (LDL) in one hour.

In earlier report (BRAHN 1940) observed a 20% rise in mean cholesterol level after a test dose of cholesterol.

There are atleast three independent predictors of risk for individuals. There are Plasma cholesterol concentration, [RAM 1986, INKELES and EISEENBERG, 1981] cigarette smoking [WISSLER, 1976], and elevated blood pressure [OBERMAN, HARLEN et al 1969]

Recently some workers have shown the existence of cell surface receptors for low density lipoprotein [GOELSTEIN et. al 1974] which explains the mechanism of plasma cholesterol control.

MOGADM. M, AHMEDS W, MENSHSH (1990) :measured fasting total serum cholesterol & lipoproteins variation of more than ± 20% in serum level of total cholesterol, low density lipoprotein cholesterol and high density liporotein cholesterol were seen in 75%, 90% and 65% of subjects respectively, on observation , 40% of subjects moved in or out of

one risk category and in 10% two categories from desirable to high risk or vice versa. These data demonstrate that random testing may fall to detect wide fluctuation in level of serum lipoprotien & therefore result in erroneous risk assignment or therapeutic intervention [ARCH INTERN MED 1990, 150 (8) 1583-5] various hypothesis have been proposed to explain atherosclerosis. Dietary fat cholesterol and raised plasma cholesterol are one of the major risk factors for atherosclerosis.

LAREN (1966) observed that reduction in dietary cholesterol however has no change in subjects above 60 year of age, when Polyunsaturated fat have been substituted for saturated fats in diet it will lower serum cholesterol but not serum triglyceride [AHRENS et al ; 1957; KINSELL et al 1952, GRUNDY, 1975]

Serum cholesterol was higher during the first 9-12 months of life in breast fat babies because breast milk is rich of cholesterol. In subsequent life there was little differences [FRIEDMAN AND GOLDBERG 1976, HUTTMAN et al 1983].

Previous study (Arora et al, 1987) showed that dietary cholesterol and fat have difinite relation to serum lipid the study was done to evaluate the changes in serum lipoprotien after ingestion of high cholesterol fat diet in selected number of subjects. It showed that the basal values of various lipoprotiens were normal at their age and rise occured in all lipoprotein after ingestion of high cholesterol fat diet.

Atherosclerosis is a diseases of large and medium sized muscular arteries and has a basic lesion : Atheroma of fibrofatty plaque consisting of raised focal plaque within the intima having a layer of lipid mainly cholesterol usually compelxed to proteins and cholesterol esters

and a covering fibrous cap (Robbins and Cotran, 1984). The possible mechanism by which HDL cholesterol decreases atherosclerosis includes :

1. Reveral of cholesterol transport from the peripheral cells to the liver for removal from the body (Miller and Miller, 1975).

2.Inhibition of LDL cholesterol uptake by cells at the LDL receptor sites.

FAT TOLERANCE TEST AND ITS IMPLICATIONS

The concept of fat/cholesterol tolerance test is not entirely new. In 1907, neuman after giving a fat load studied the quantitative lipid changes in from the of chylomicron count after a fat load.

Introduction of isotopes, revolutionsied the study of lipid metabolism. Brekowitz (1963). Pointed out that radioactive fat tolerance is a better index for determining the functional state of lipid metabolism.

If atherosclerosis is a post prandial phenomenon then premature CAD must be common in hyperchylomicronemic states. However, in familial lipoprotein lipase deficiency enormous quantities of chylomicrons accumulate in plasma, but accelerated atherosclerosis has not been reported (Fredrickson et al, 1970).

HDL levels are lower in obese individual than in non obese controls (Wilson et al, 1972 : Carlson et al.1975 and Glueck et al). During the course of weight loss, an increase in HDL cholesterol concentration has been reported to occur in association with reduction in VLDL and total

triglycerides concentration (Wilson et al, 1972). But in other studies HDL cholesterol showed either no change or a reduction (Windholm et al, 1978, Thompson 1979, Howard, 1979).

Serum cholesterol levels have been reported to be higher in postmenopausal compared with premenopausal women of same group in several population in the united state and North Europe (Halberg et al, 1957 ; hjortland et al, 1976 ; Lindquist et al, 1980). A rise in serum cholesterol with menopause has also been reported in Japanese women (Shibata et al. 1963).

The women using oral contraceptive that are higher in estrogen and low in progestin contest had significant high concentration of HDL cholesterol than those not using the hormones (Glose et al, 1974 ; Hironen et al, 1981 ; and Larsson Cohn et al. 1979).

The basic defect in reduced number of LDL receptors.In normal person about 45 poercent of the plasma LDL pool is removed from the plasma daily by the receptors where in familial hypercholesterolemia heterozygotes this value is 25-30 percent and in homozygotes it is about 15 Percent. The receptor deficiency results in accumulation of LDL into the plasma leading to raised level and premature atherosclarosis.

The Fremingham's study (1976) showed that in men and women 35-44 years of age, serum cholesterol levels 265 mg/dl or more have five times higher' risk of IHD than are levels 220 mg/dl or less. The most striking association with atherosclerosis (AS) and IHD is with elevated levels of LDL (Weiss et al, 1972). But Hyperlipidemia with inereased

concentraduon of VLDL also appears to increase the risk, In contrast serum levels of HDL are inversely related to risk (Heirs et al 1980).

CONTROL OF PLASMA CHOLESTEROL LEVEL BY LDL RECEPTORS

A decade of intense investigation has established a central role for lipoprotein receptors in regulating plasma cholesterol. Operationally, the IDL/LDL receptor system can be considered the primary transport mechanism for endogenous cholesterol. LDL are generated in the plasma by the degradation of intermediate density lipoprotein (IDL) Generated LDL is removed relatively solowly rom plasma by binding to LDL receptors in the liver and extra hepatic tissues (Kita et al, 1982). However, the precise distribution of these receptors in man is unknown.

REGULATION OF HEPATIC LDL RECEPTORS

Hepatic LDL receptors are suppressed whenever the liver content of cholesterol increases or its demand for cholesterol is reduced . Thus receptor suppression occurs when a high cholesterol diet is consumed (Hui et al, 1981) or when bile acids are infused (Angelin et al, 1983). Conversely, LDL receptors increase when hepatic cholesterol synthesis is blocked by drugs compactin or mevinolin (Goldstein et al, 1982 and Bilheimer et al, 1983).

LIPID METABOLISM

EXOGENOUS PATHWAY

The chylomicrons, large triglycerides rich particles are produced in the intestine from dietary fat. Hence they are normally not

present in plasma after fast of 12-14 hours. They are catabolized by lipoproteins lipase (LPL) and hepatic lipase (HL) to form chylomicrons remnants, triglycerides form free fatty acids (FFA). Apo E facilitates the uptake of these remnants while Apo C-III inhibits it.

ENDOGENOUS PATHWAY

VLDL synthesis occurs in liver and is incresed in obese persons and is inhibited by the uptake of chylomicrons remnants. VLDL, triglyceides and phospholipids are hydrolyzed by lipoprotein lipase and hepatic lipases. During this apo E and apo C of VLDL is transferred to HDL while apo B-100 remains within. Thus the end product of VLDL catabolism are LDLs.

LDLs are major cholesteol carrying lipoproteins in normal plasma in humans and most of it coms from VLDL catabolism while some are synthesized directly (in subjects of homozygous familial hypercholesterolemia). The major protein of LDL is apo B-100 is catabolized in various cell types by receptor dependent as well as receptor independent mechanism. LDL when degraded in cell results of from free cholesterol which inturn inhibits the enzyme (3 hydrxy, 3 methylglutaryl coenzyme A and reductase) producing it.

Direct HDL production occurs in liver and intestines and also derived from chylomicrons and VLDL catabolism. Moreover, HDL serves as acceptor of lipid especially free cholesterol. Apo I and II are major protein in HDL. Hepatic lipases metabolise HDL phospholipids and triglycerides and liver and kidney are major sites for catabolism.

CHYLOMICRONS

It is the largest of the lipoproteins originating from the gut mainly composed of triglyceride and transport dietary triglyceride and cholesterol from gut to site of metabolism or storage. In post prandial state it is detacted by " creaming in the cold".

Dietary fat is broken down to free fatty acids and monoglycerides in intestine which then enterintestinal villi in jejunum reconstituting into triglyceride. In the cells of jejunum cholesterol is esterified to cholesteryl ester (Oleate). The triglyceides are then complexed with Apo B-48. apo AI & II A-IV within intestinal wall. The chylomicrons enter systemic circulation via lymphatics, Apo E and apo C proteins are added in lymph or blood. Chylomicrons are rapidly cleaned from blood by lipoprotein lipases and results in formation of partially degraded chylomicrons particles called remanants which are taken up by liver.

Chylomicrons in fasting state is abnormal and has been postulated that prolonged clearance of dietary remnant particles could be damaging to vascular endothelium and may predispose to atherosclerosis.

VLDL

It is endogenously produced lipoprotein (in liver) and contains apo B-100. It's synthesis is increased in obesity, alcohol use and diabetes, nephrotic syndrome and hypothyroidism. Its function is to transport cholesterol and endogenously produced triglyceride. Clefsky et al (1976) noted biphasic plasma triglyceride curve. An initial peak occureed 1-3 hours after feeding was accounted by increase in chylomicrons levels in more than 98% and second peak after 4-7 hours accounted for rise in VLDL level in 82%.

IDL

It is formed from metabolism of VLDL of which roughly half is metabolized in mass and remaineder half in converted to LDL. The elevation of IDL is also thought to predispose for atherosclerosis.

LDL

It is produced from VLDL in plasma, and LDL supplied cholesterol to extrahepatic parenchymal cells, as adrenal cortical eells, lymphocytes, muscle cells etc. Thus Goldstein (1977) hypotesized the concept of LDL receptors and has been confirmed by many laboratories. These receptors are over cell surfaces to which LDL binds and by endocytosis. it is digested by lysosomes literating cholesterol for membrane synthesis and precurrs for steroid harmone synthesis. Liver uses LDL for synthesis of bile acids and free cholesterol secreted in bile. Diet high in fat and cholesterol causes elevation of LDL but varies in man.

Age related difference in rise of LDL was demonstrated by Arora and Gupta G (1987). They found out that rise of STC after feeding high fat breakfast for one week was much more pronounced in young voluntees (20-30 years) with major portion of rise contributed by HDL. Contrary in other persons the rise of STC was less marked and LDL mainly contributes to it.

HDL

It is produced in gut, by liver and also by peripheral catabolism of chylomicrons and VLDL. They are reservoirs for apolipoproteins. Some investigators have proposed that HDL facilitates cholesterol removal from

cells particularly of reticuloendothelial system (Schnitz and Robenek et al, 1985). It is thus termed reverse endocytosis.

HDL is subdivided into several fractions in which HDL_2 and HDL_3 are important and best studied. HDL_2 are lage and more lipid rich than HDL_3. Concentration of HD_2 is higher in woman than in man and are increased by oestrogen or physical activity (Exercise). Alcohol also increases both HDL_2 and HDL_3. Factors in which HDL lowers are hypertrigly ceridemia, cigarette smoking. Exogenous androgen administration lowers HDL levels in man (Furman et al, 1967). A drop in HDL level is seen in males at around the time of puberty (Beagtehole et al, 1980) and has been related to degree of sexual matuation (Frerich et al , 1978 and Morrison et al, 1979). Transient increase of HDL2 has been reported at time of ovulation (Barclay et al, 1965). No changes in HDL is found during pregnancy.

HDL level changes with age. In males levels are stable uptill puberty after which there is decline followed by stable levels in adulthood until 55-60 years where there is increase and then a plateau in older age group. In females there is a small lineare increase in levels from childhood to about 60 years.Reduction in obesity by mild exercise programme resulted in no increase in HDL cholesterol while drop in HDL levels a are found in those with caloric restriction in absence of exercise.

Effect of Dietary cholesterol on lipid metabolism

Additions of dietary cholesterol has been known to increase plasma cholesterol levels and induce arteriosclerosis in experimental

animals. Subsequently cholesterol rich diets have regularly caused hypercholesterolemia, atherosclerosis and even myocardial infarction in a large number of experimental animals including primates (Taylor et al, 1950 ; Armstrong et al, 1967 ; 1970). A decisive effect of cholesterol in the diet of man upon the serum lipid levels was clearly demonstrated in series of metabolic ward experiments being carried out in normal voluteers (Beveridge et al, 1960 ; Conner et al, 1961 ; 1964).

Absorbed cholesterol is transported from the gut in chylomicrons, largely as esterified cholesterol and reaches a peak concentration in plasma 48 hours after meal. Chylomicrons are converted into chylomicron remants after the action of lipoprotein lipase in peripheral tissues. The cholesterol of remnants contributes its mass to the tyotal body pools of cholesterol (Bhattacharya et al, 1976).

The increased cholesterol uptake may :-

1. Inhibit new cholesterol synthesis.

2. Increase esterol excretion in the bile as bile acids or as cholesterol it self.

3. Increase excretion of cholesterol from the liver as nearly synthesized lipoproteins primary VLDL.

Effect of carbohydrates of plasma lipid levels

Carbohydrates induction occure when 70-80% of calories are supplied by carbohydrate. In carbohydrate induction plasma triglyceride level start to increase transiently plasma triglyceride level start to increase

transiently (Ahrens et al, 1961). Individuals on high carbohydrate died have insignificant rise in triglyceride level and low incidence of atherosclerosis (Conner and Conner 1972)

Effect of calories on plasma lipid level

The consumption of high calories (more than basal requirement) is associated with obesity in which cholesterol synthesis is increased (Dennion and Grundy, 1975). Carlson et al (1975) and Garrison et al (1980) reported the elevated levels of VLDL, LDL and reduction in HDL is associated with obesity. These consequenes are due to increased synthesis of VLDL, triglyceride and decreased clearance of VLDL, triglyceride and decreased clearance of VLDL from after high calories supplementation.

Effects of Dietary fat on lipid metabolism

The amount and kind of fat in diet has a well documented effect upon plasma lipid concentration (Ahrens et al, 1957 ; keys et al, 1957). A fatty meal will result in the productions of large number of chylomicrons. The effects of dietary fat upon plasma concentration depends on the type of fat consumed. Long chain saturated fatty acids are not essential nutrients and may be synthesized in the body from acetate. Dietary saturated fatty acids increases the LDL concentration (Ahrens et al, 1957 ; and Keys et al, 1957). All the animal fats except fish are highly saturated.

These are no essential fatty acids and have no effect on plasma lipid. They are readily synthesized in body. Polyunsaturated fatty

acid (prostaglandin precursors) are important constituents of cell membrane. They are rich in vegetables oils, fish and shellfish. These polyunsaturated fatty acids serve as substrate for the formation of different prostaglandin and concentrated in nervous tissue retina, spermatozoa, adrenal glands and many other organs. They depress plasma cholesterol and LDL concentration. Polyunsaturated and saturated fatty acid ratio known as P/S value. The fats having P/S value more than 2 are generally recongnised as hypocholesterolemic.

The mechanism of lipid lowering effect of polyunsaturated fatty acid is not clear. Sheoherd et al (1980), Turner et al (1980) postulated that, it increases in the clearance and decrease synthesis of LDL in normal individual.

AIMS OF STUDY

AIMS OF STUDY

1. To find out the basal cholesterol and other lipoprotien in Indian population.

2. To find out the day- to - day variation in serum cholesteol and other lipoprotein in healthy indian population.

MATERIAL AND METHOD

Material and Methods

Present study consisted of 32 healthy subjects their age ranged from 20-40 years with mean age of 29.62 ± 6.11 years. These all subject were selected from admitted patients in wards of MLB Medical college hospital Jhansi and few of them were patiant's attantend

Subjects were divided in two groups on the basis of age and sex.The age range were 22-40 year with Mean age 28.76 ± 5.85 yeas of 17th male subjects and 15th female age range 20-40 years with Mean age 30.6 ± 6.44 years.

Informed consent taken from each subject. A detailed history, thorough clinical Examination and relevant investigations e.g. Blood sugar, Bl urea, TLC, DLC, Hb% ESR and urine protien were done. All subjects were enquired about their dietary habit, socioeconomic status. Use of smoking, tobacco chewing and Alcohol Consumption were not allowed during whole period of study.

Detailed family history was enquired in every subjcts regarding Diabetes Mellitus, Inschaemic heart desese : hypertension, obesity etc.

Design (Protocol) of Test

The subject were asked to take their dinner at 8. Pm. on Previous night and not to take anything except water after this, next morning fasting blood samples (2 ml) was taken serum was separated from blood within 4 hours by centrifugation & following test were performed ;

Mode of estimation

(1) Serum total cholesterol (STC) :-

STC estination was done by kit : with the help of one slep method supplied by ethnor India limited.

Procedure :-

Three test tubes are taken and labelled as Test (T), standard (S) and Blank (B) and then :

	Test (T)	**STD** (S)	**Blank** (B)
Ortho cholesterol reagent	4 ml	4 ml	4 ml
Seum	29 ul	-	-
Cholesterol standard (250 mg%)	-	20 ul	
Distilled water	-	-	20 ul

Mix contents of each test tube simultaneously for 10 seconds and immediately place them in a boiling water bath for exactly 45 seconds followed by cooling with running tap water or cold water for 5 minutes. Dry the exterior of tube mix their contents.

Measure optical density (OD) of each solution at 560 nm (Range560 to 600 nm). Set blank at calorimetric zero and calculation was done as

$$\text{Cholesterol concentration of test samples (mg\%)} = \frac{\text{OD (T)}}{\text{OD (S)}} \times 250$$

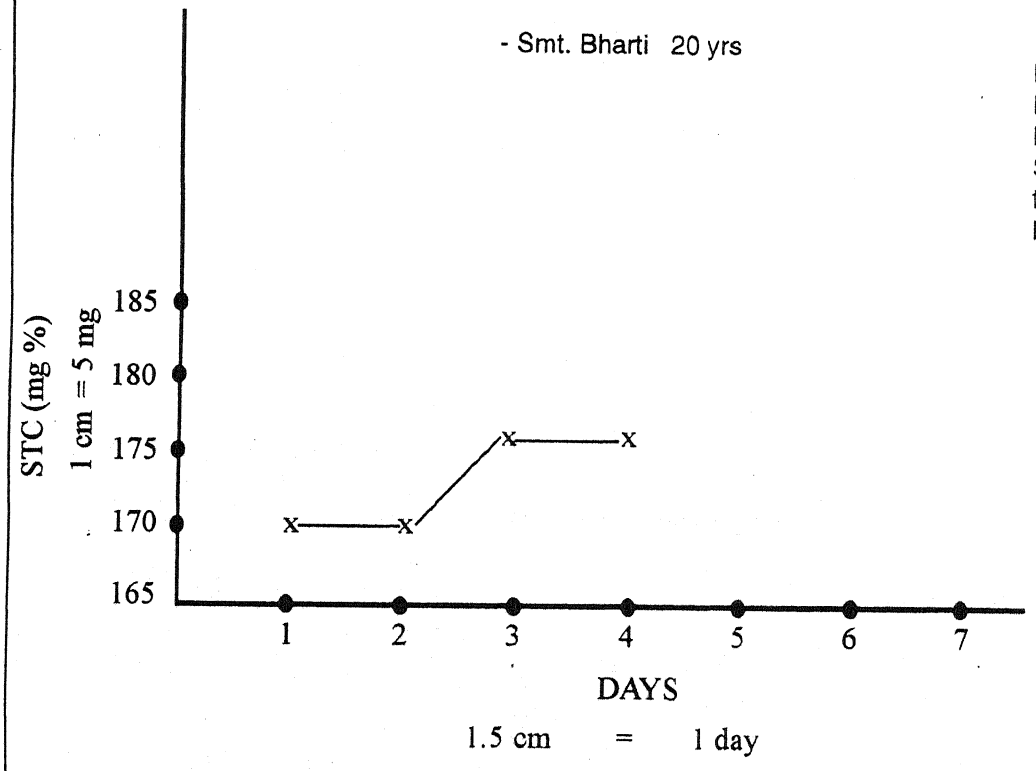
(Cholesterol mg/dl or mg% / 38.7 = mmol /l).

normal expected values of Cholesterol = < 200 mg /dl.

OBSERVATION

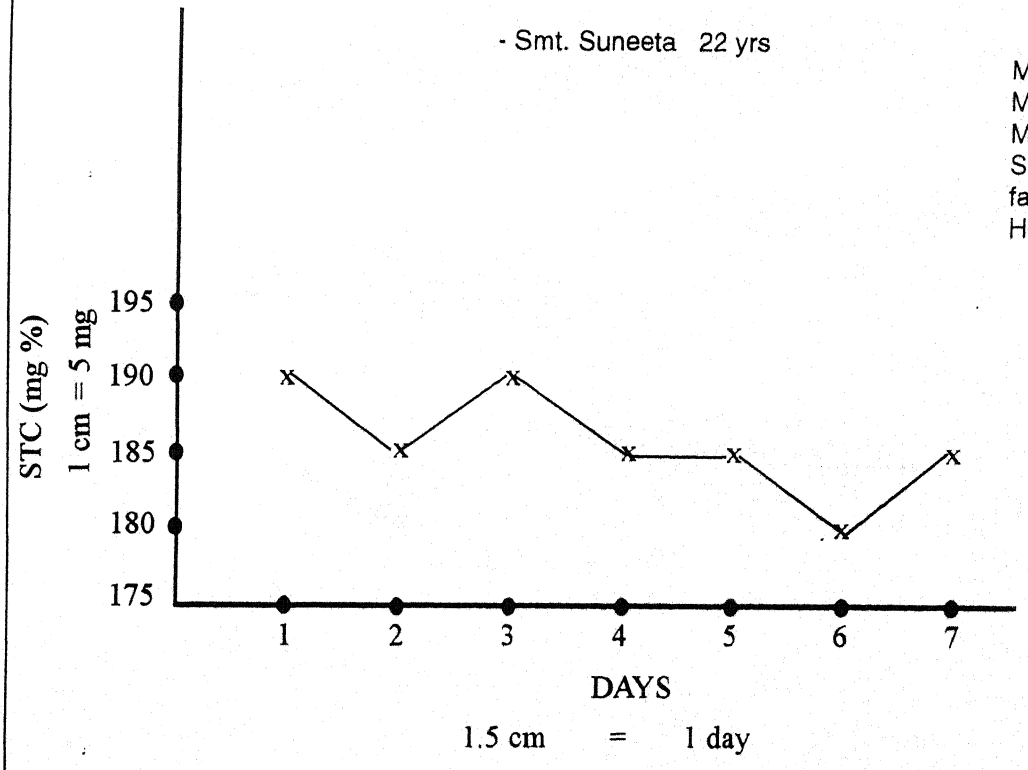
- Smt. Bharti 20 yrs

Max. value = 178 mg
Min. value = 171 mg
Mean Value = 183 ± 3.10
Smt. Bharti has/day variation in fasting STC
However within normal range

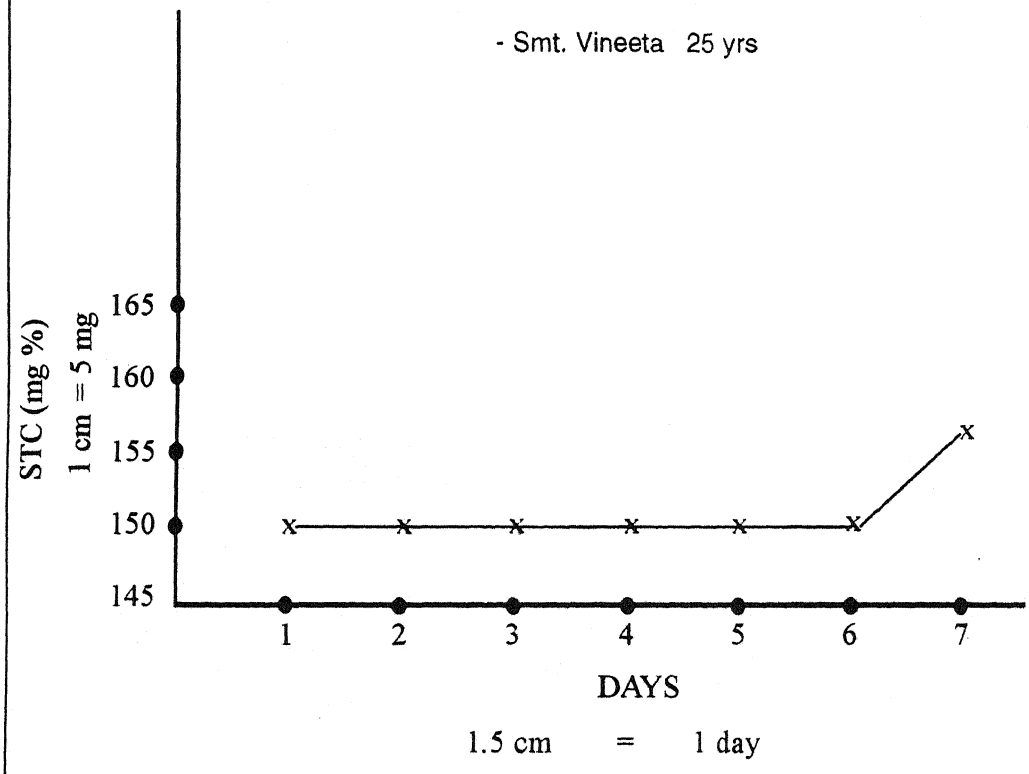


- Smt. Suneeta 22 yrs

Max. value = 190 mg
Min. value = 180 mg
Mean Value = 186.71 ± 3.45
Smt. Suneeta has/day variation in fasting STC
However within normal range

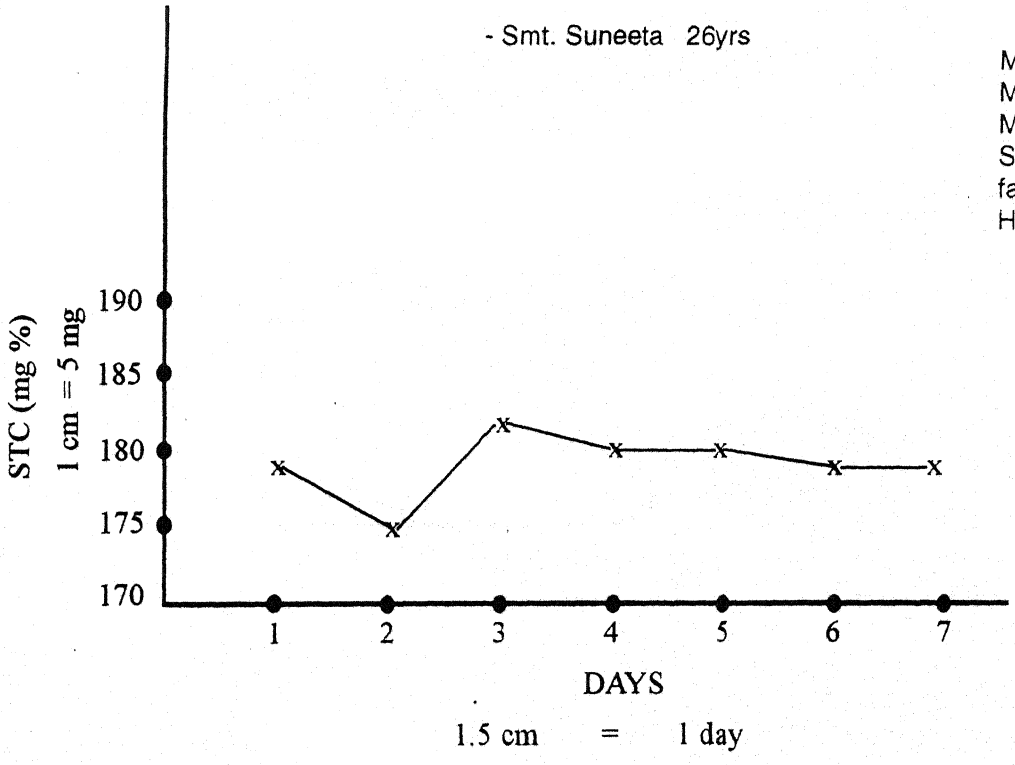


- Smt. Vineeta 25 yrs



Max. value = 157 mg
Min. value = 150 mg
Mean Value = 151 ± 2.64
Smt. Vineeta has/day variation in fasting STC
However within normal range

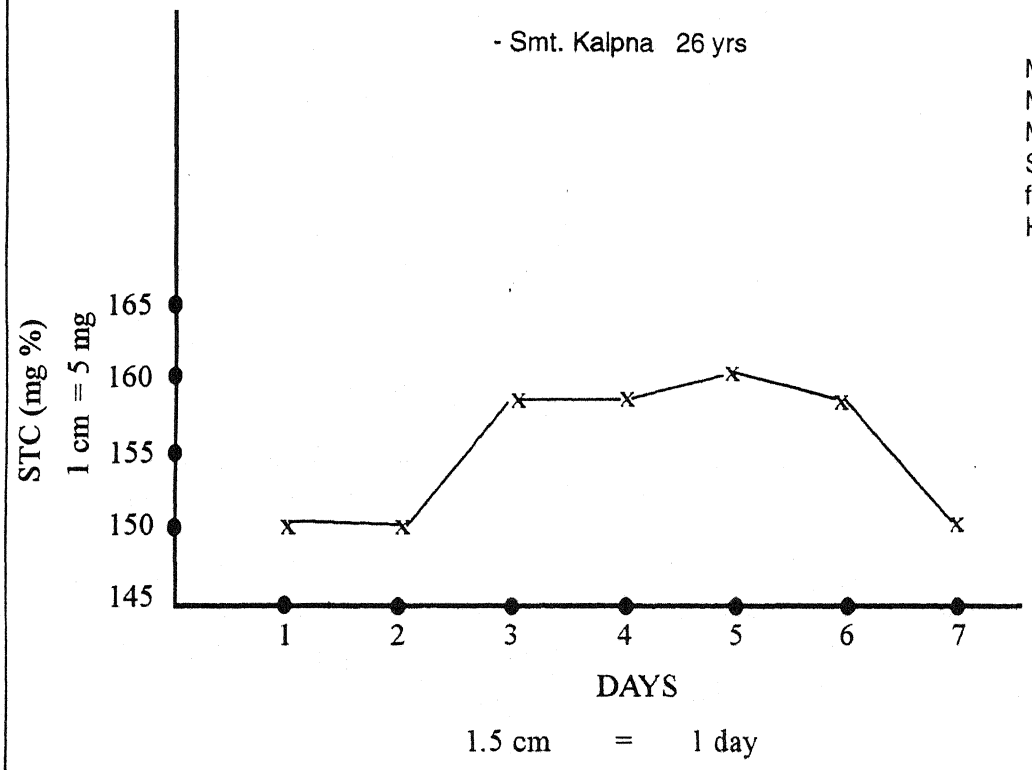
- Smt. Suneeta 26yrs



Max. value = 182 mg
Min. value = 175 mg
Mean Value = 178.71 ± 2.21
Smt. Suneeta has/day variation in fasting STC
However within normal range

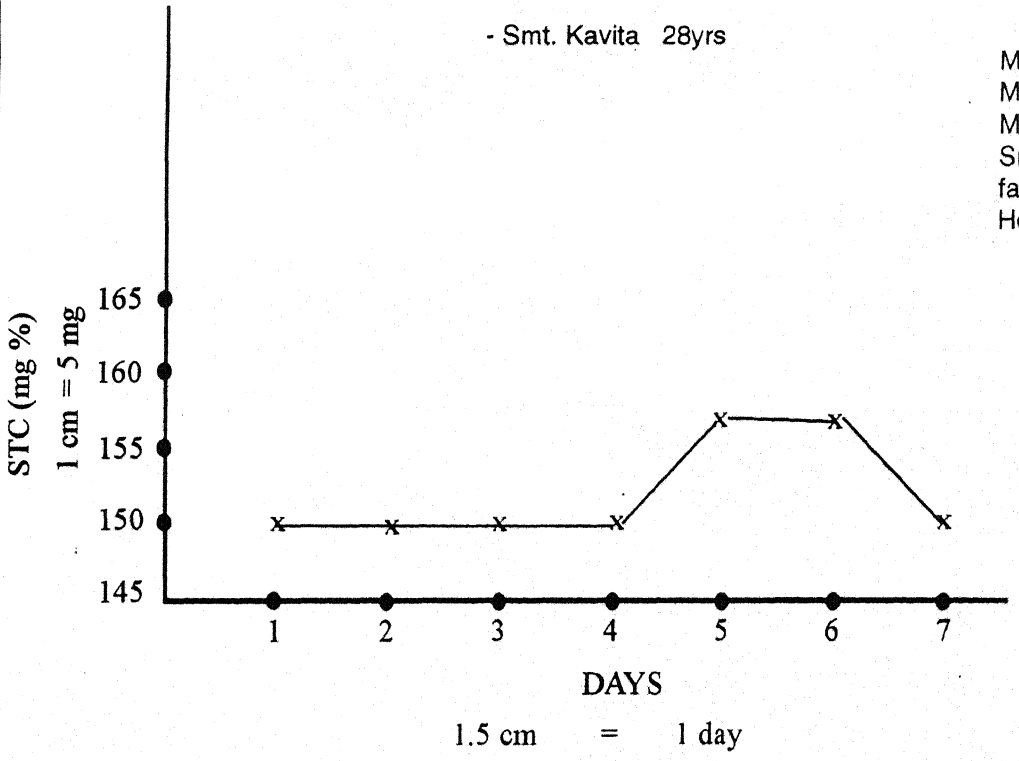
- Smt. Kalpna 26 yrs

Max. value = 160 mg
Min. value = 150 mg
Mean Value = 154.42 ± 4.27
Smt. Kalpna has/day variation in fasting STC
However within normal range

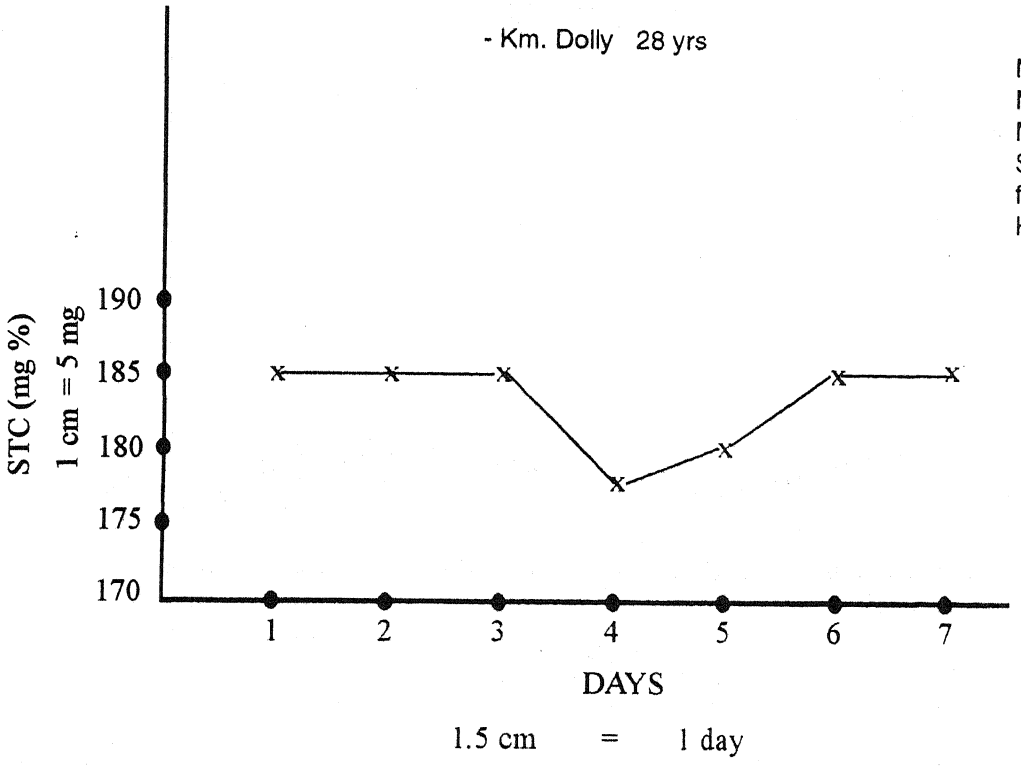


- Smt. Kavita 28yrs

Max. value = 157 mg
Min. value = 150 mg
Mean Value = 152 ± 3.41
Smt. Kavita has/day variation in fasting STC
However within normal range

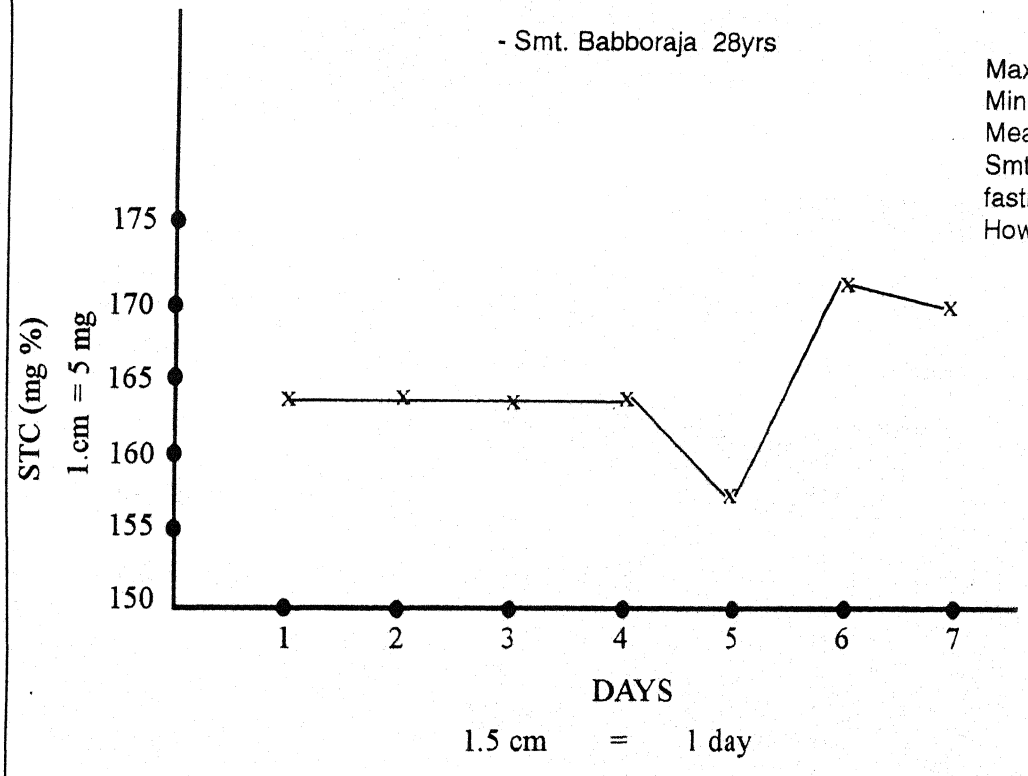


- Km. Dolly 28 yrs



Max. value = 185 mg
Min. value = 177 mg
Mean Value = 183.14 ± 3.28
Smt. Dolly has/day variation in fasting STC
However within normal range

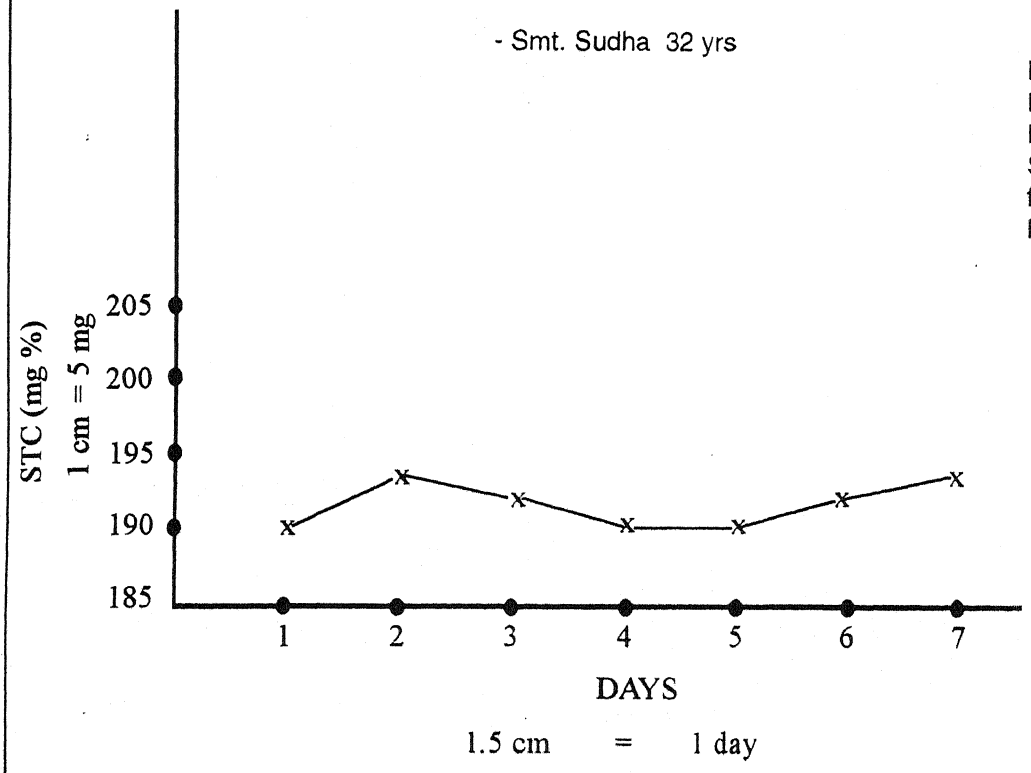
- Smt. Babboraja 28yrs



Max. value = 171 mg
Min. value = 157 mg
Mean Value = 164.81 ± 4.63
Smt. Babboraja has/day variation in fasting STC
However within normal range

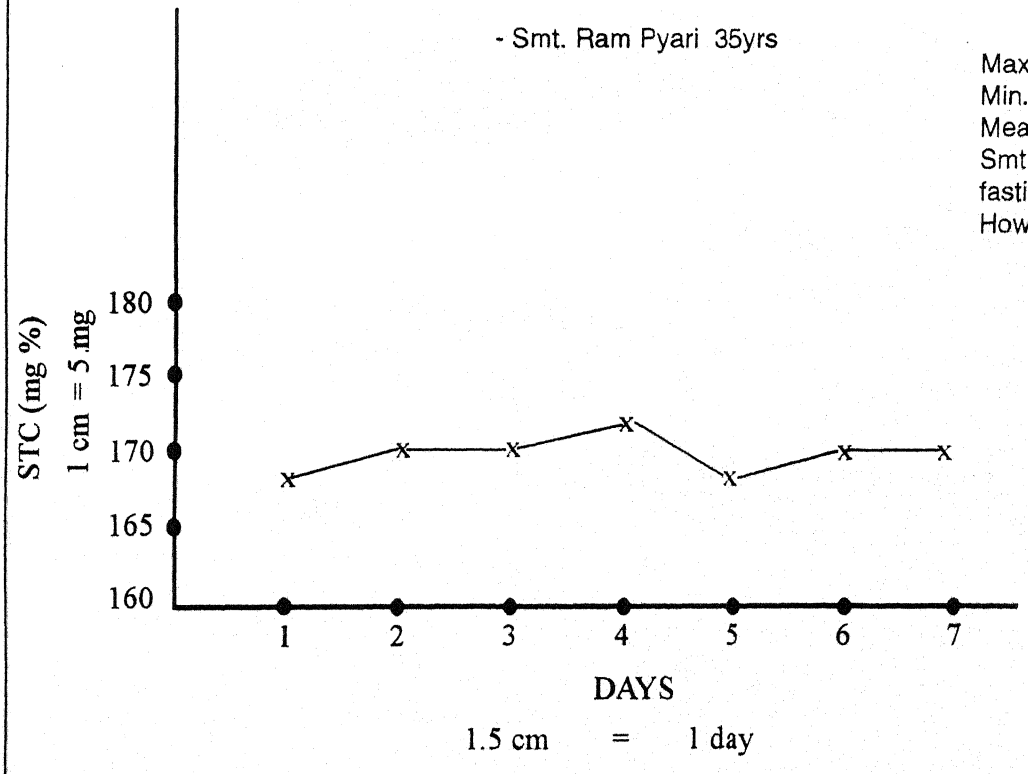
- Smt. Sudha 32 yrs

Max. value = 194 mg
Min. value = 190 mg
Mean Value = 191.71 ± 1.79
Smt. Sudha has/day variation in fasting STC
However within normal range



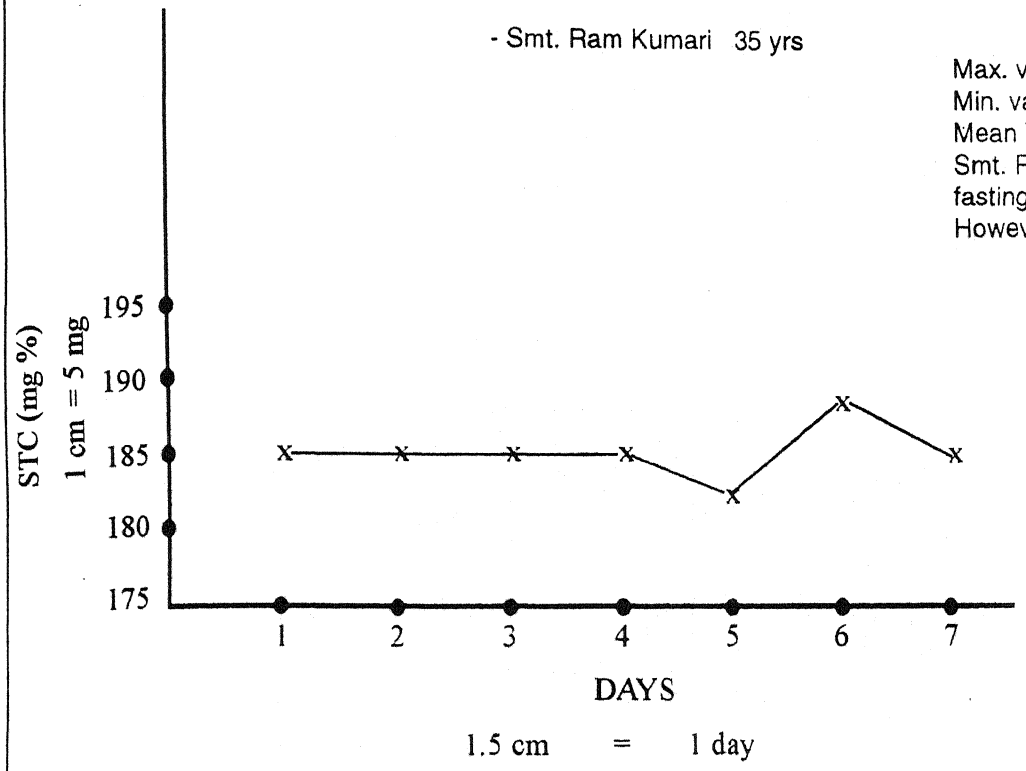
- Smt. Ram Pyari 35yrs

Max. value = 172 mg
Min. value = 168 mg
Mean Value = 169.71 ± 1.38
Smt. Ram Pyari has/day variation in fasting STC
However within normal range



- Smt. Ram Kumari 35 yrs

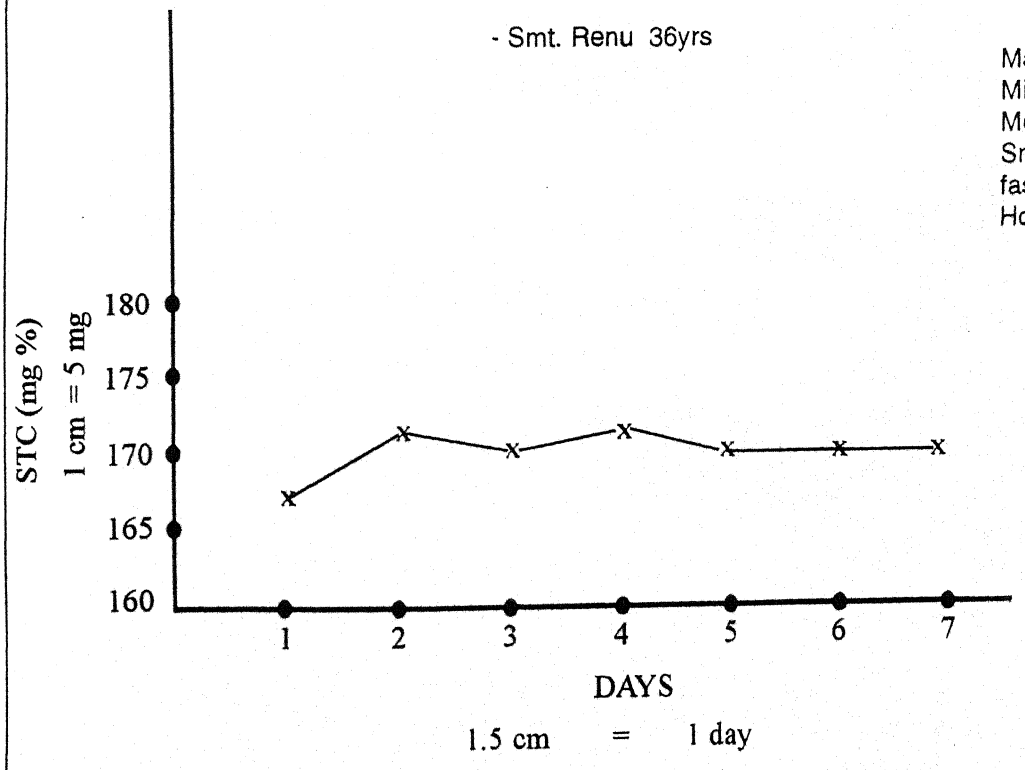
Max. value = 188 mg
Min. value = 182 mg
Mean Value = 185 ± 1.73
Smt. Ram Kumari has/day variation in fasting STC
However within normal range



1.5 cm = 1 day

- Smt. Renu 36yrs

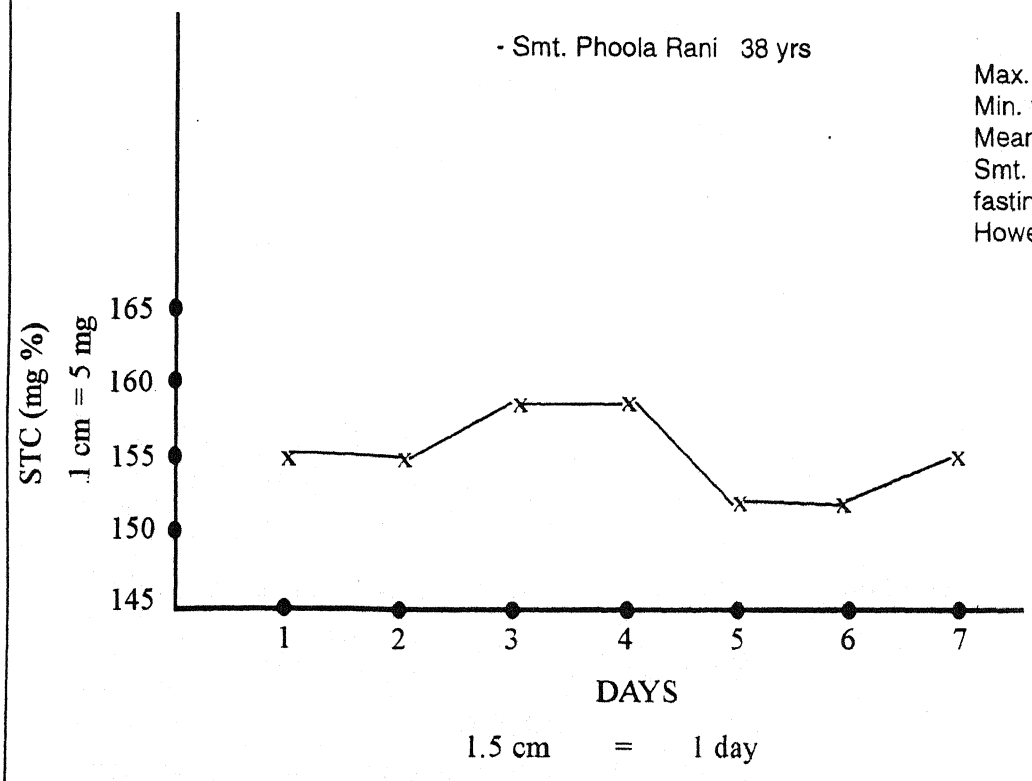
Max. value = 171 mg
Min. value = 167 mg
Mean Value = 169.85 ± 1.34
Smt. Renu has/day variation in fasting STC
However within normal range



1.5 cm = 1 day

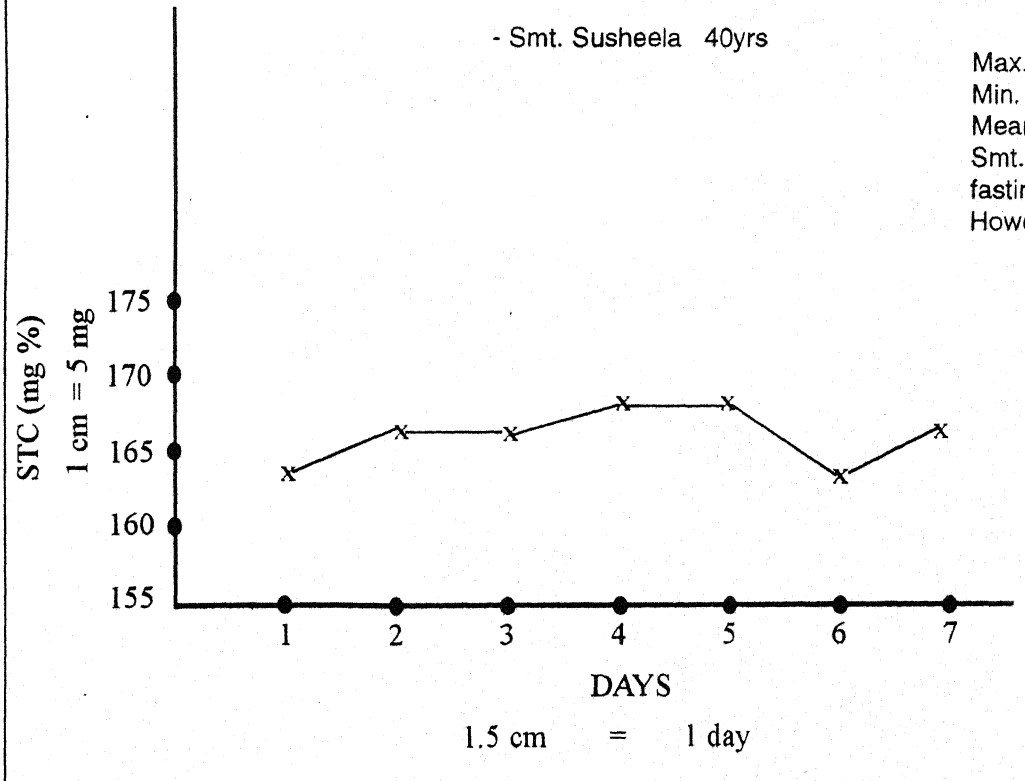
- Smt. Phoola Rani 38 yrs

Max. value = 157 mg
Min. value = 152 mg
Mean Value = 154.71 ± 2.05
Smt. Phoola Rani has/day variation in fasting STC
However within normal range



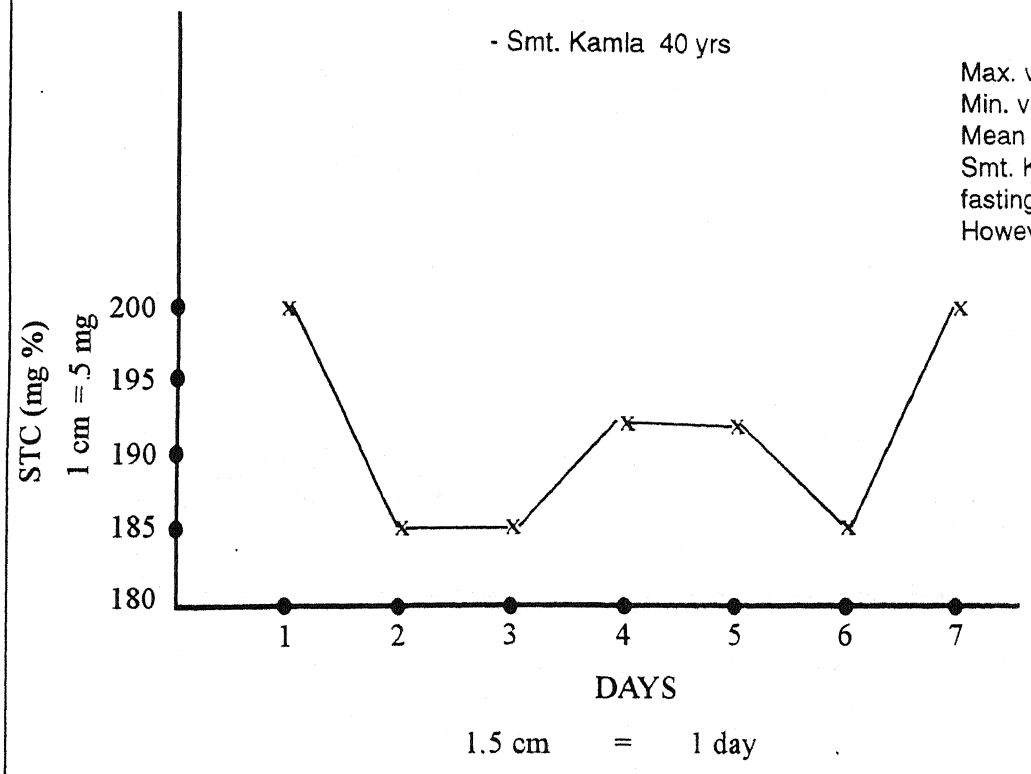
- Smt. Susheela 40yrs

Max. value = 168 mg
Min. value = 164 mg
Mean Value = 166 ± 1.63
Smt. Susheela has/day variation in fasting STC
However within normal range



- Smt. Kamla 40 yrs

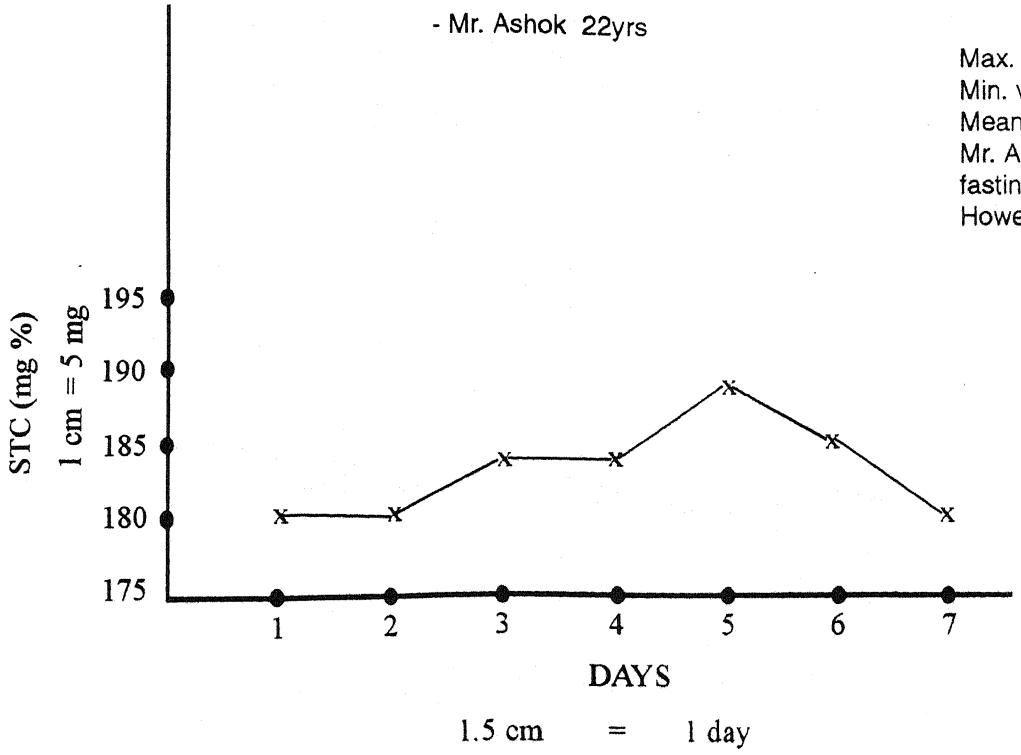
Max. value = 200 mg
Min. value = 185 mg
Mean Value = 191.28 ± 6.72
Smt. Kamla has/day variation in fasting STC
However within normal range



1.5 cm = 1 day

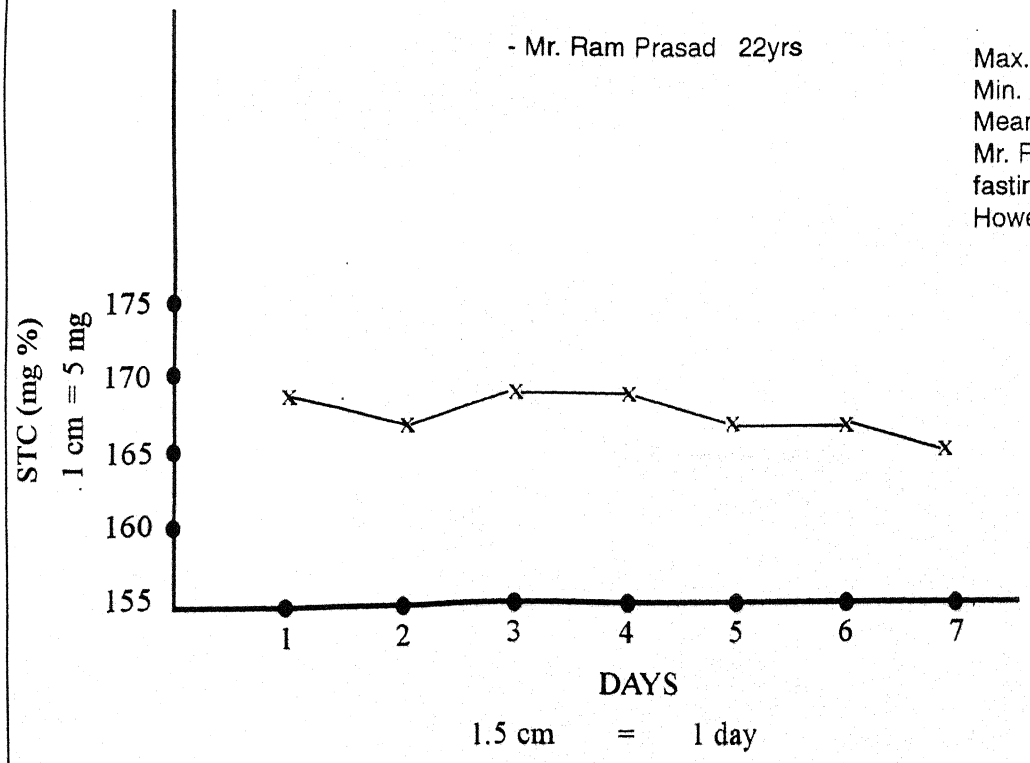
- Mr. Ashok 22yrs

Max. value = 188 mg
Min. value = 180 mg
Mean Value = 183 ± 3.10
Mr. Ashok has/day variation in fasting STC
However within normal range



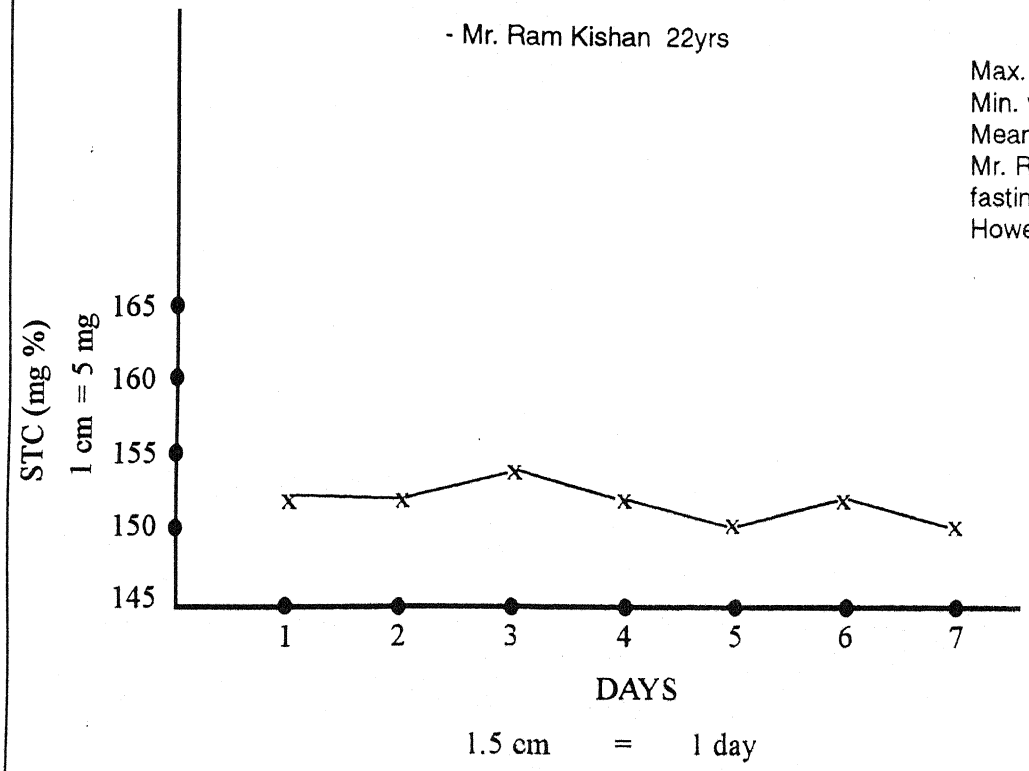
- Mr. Ram Prasad 22yrs

Max. value = 168 mg
Min. value = 165 mg
Mean Value = 166.71 ± 1.25
Mr. Ram Prasad has/day variation in fasting STC
However within normal range



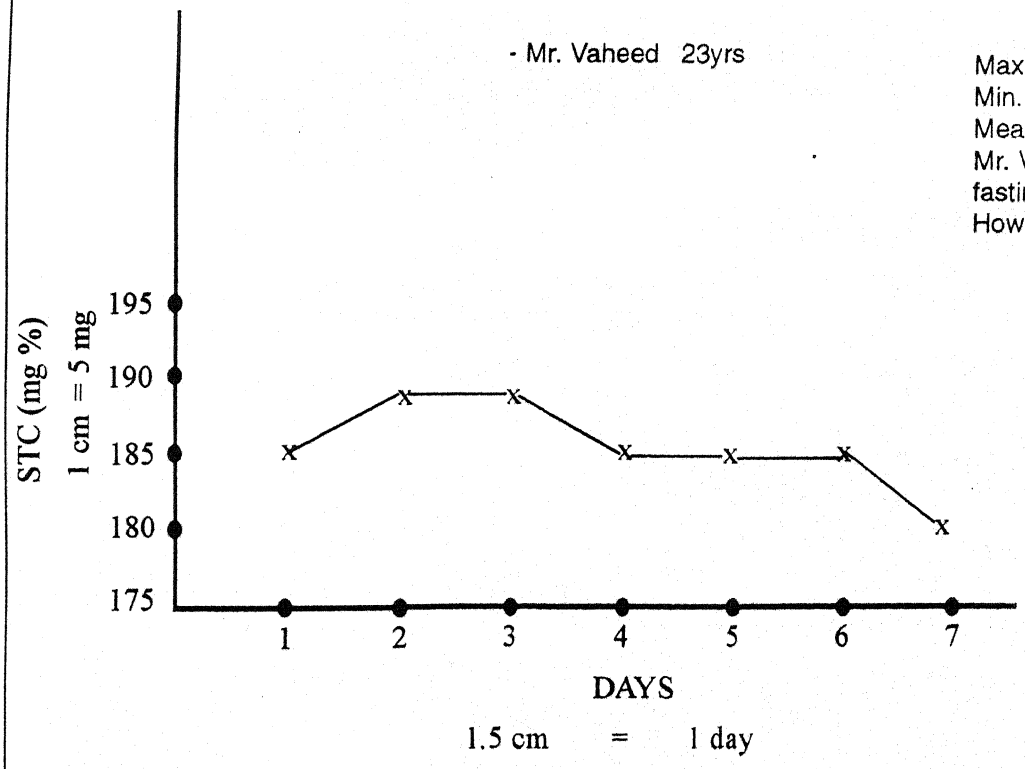
- Mr. Ram Kishan 22yrs

Max. value = 154 mg
Min. value = 150 mg
Mean Value = 151.71 ± 1.38
Mr. Ram Kishan has/day variation in fasting STC
However within normal range

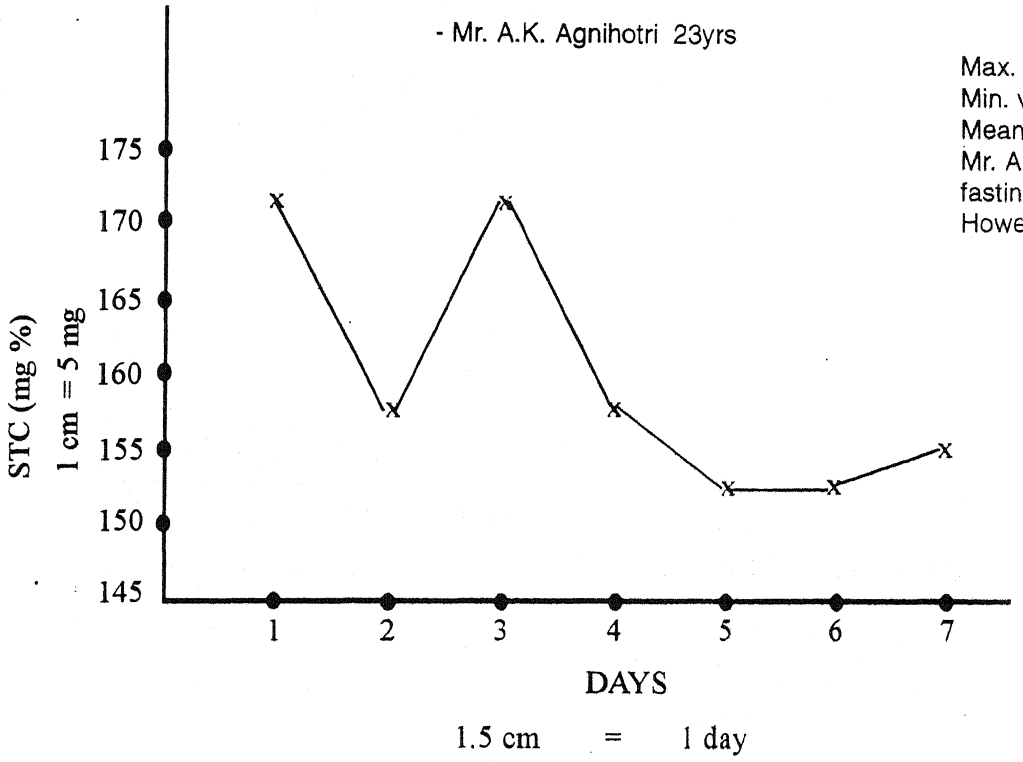


- Mr. Vaheed 23yrs

Max. value = 188 mg
Min. value = 180 mg
Mean Value = 185.14 ± 2.67
Mr. Vaheed has/day variation in fasting STC
However within normal range

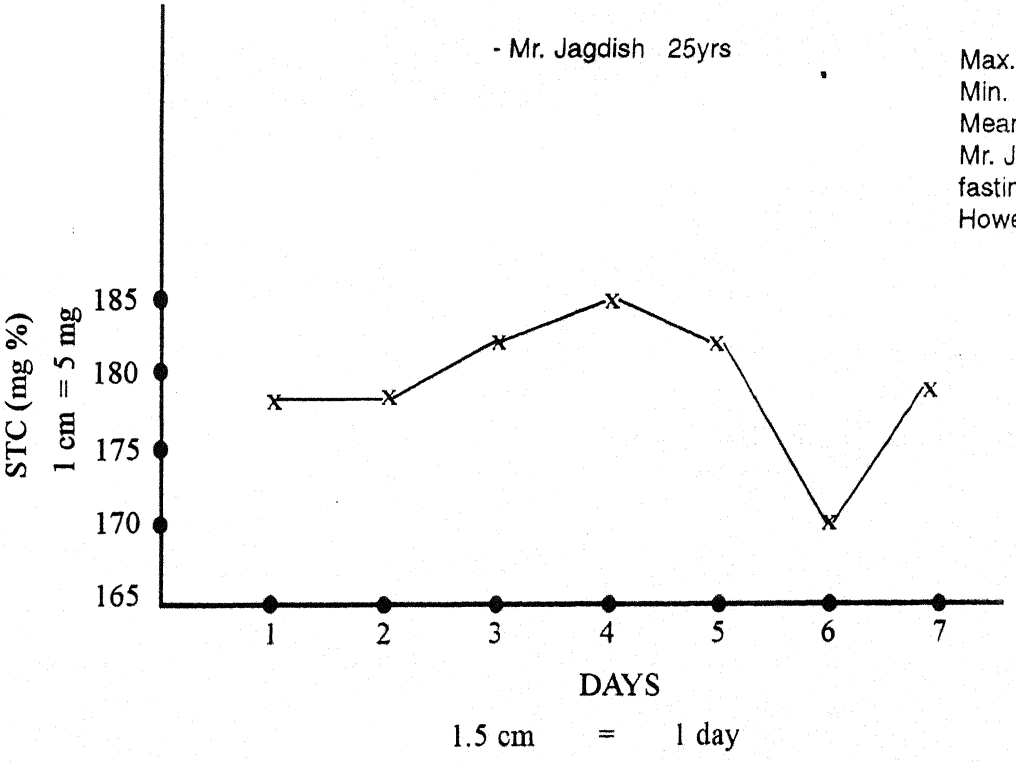


- Mr. A.K. Agnihotri 23yrs



Max. value = 171 mg
Min. value = 152 mg
Mean Value = 159.28± 8.26
Mr. A.K. Agnihotri has/day variation in fasting STC
However within normal range

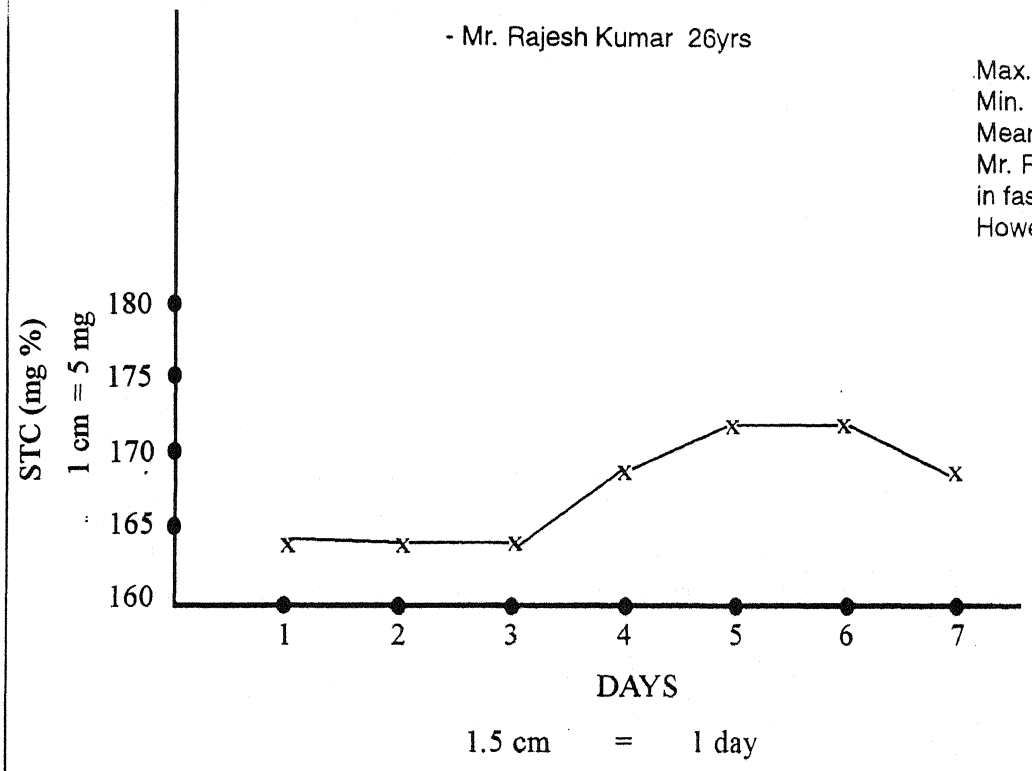
- Mr. Jagdish 25yrs



Max. value = 185 mg
Min. value = 170 mg
Mean Value = 176.71 ± 4.88
Mr. Jagdish has/day variation in fasting STC
However within normal range

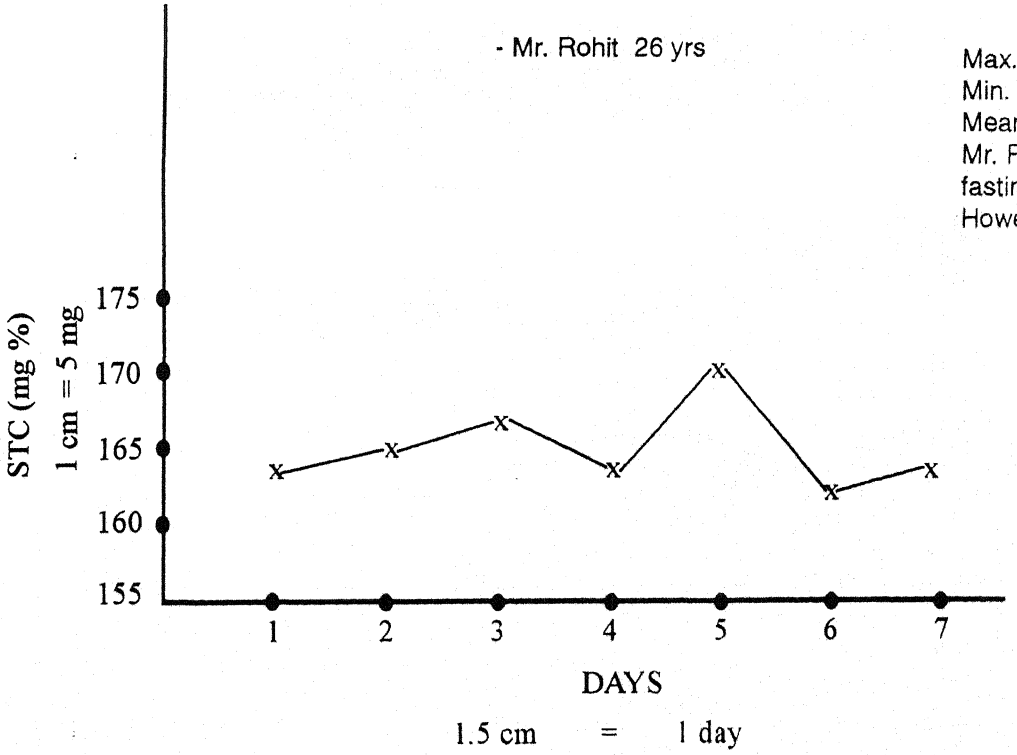
- Mr. Rajesh Kumar 26yrs

Max. value = 171 mg
Min. value = 164 mg
Mean Value = 167.14 ± 3.18
Mr. Rajesh Kumar has/day variation in fasting STC
However within normal range



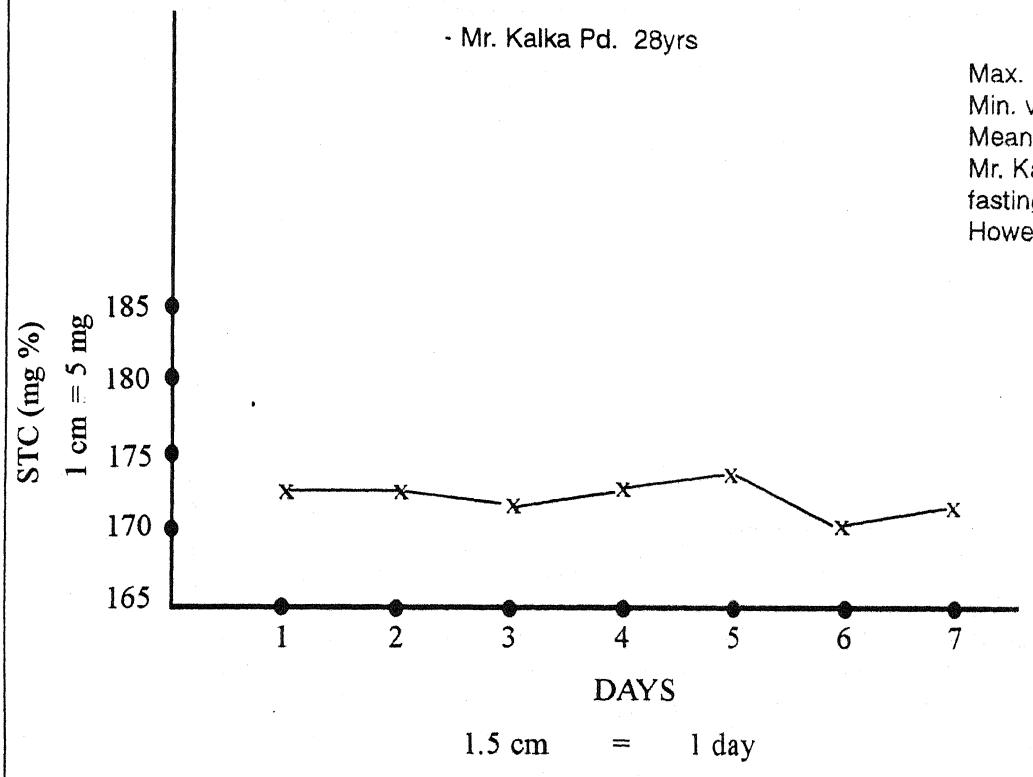
- Mr. Rohit 26 yrs

Max. value = 170 mg
Min. value = 162 mg
Mean Value = 165.14 ± 2.51
Mr. Rohit has/day variation in fasting STC
However within normal range



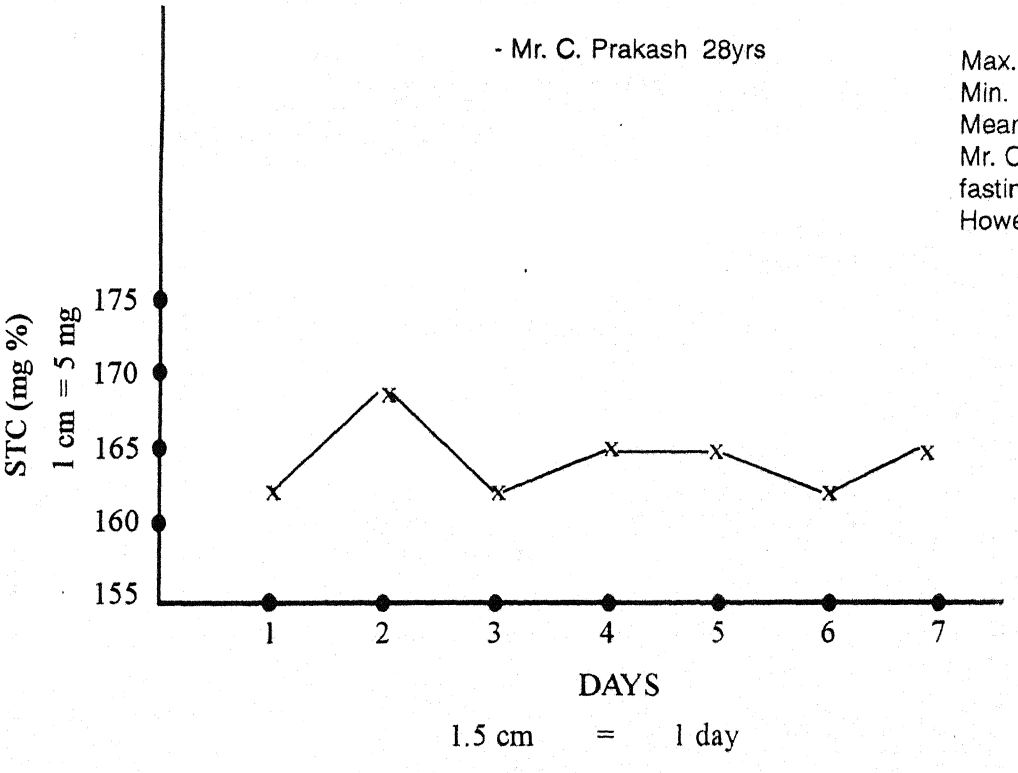
- Mr. Kalka Pd. 28yrs

Max. value = 174 mg
Min. value = 170 mg
Mean Value = 171.71 ± 1.25
Mr. Kalka Pd. has/day variation in fasting STC
However within normal range



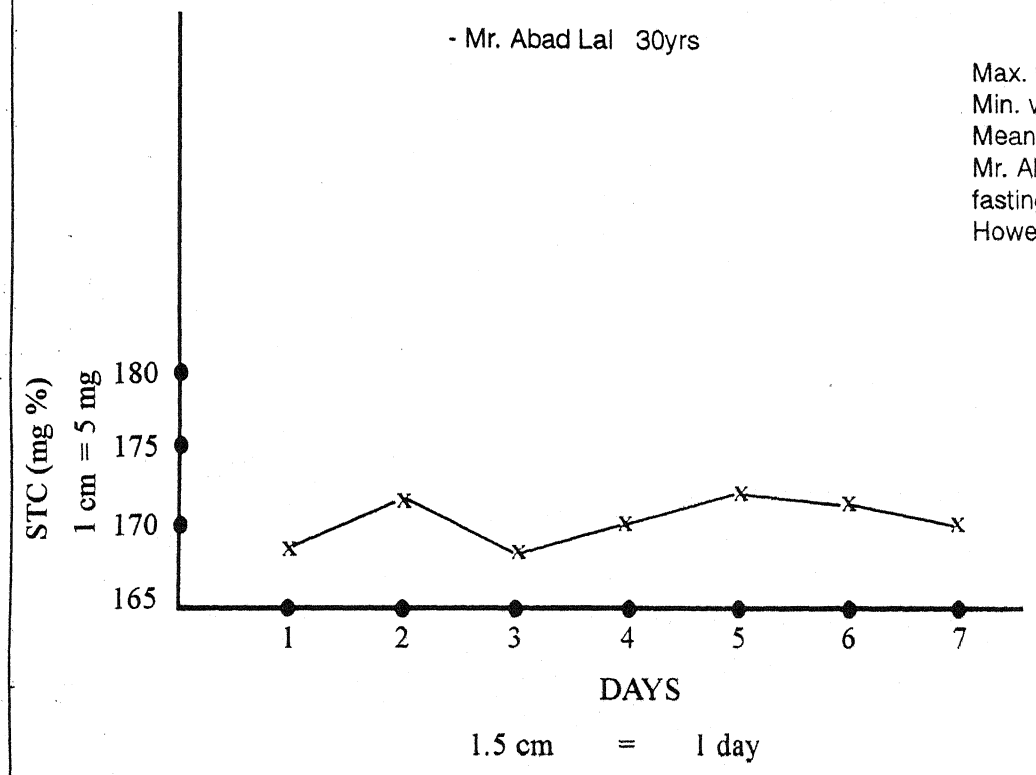
- Mr. C. Prakash 28yrs

Max. value = 167 mg
Min. value = 162 mg
Mean Value = 164 ± 2
Mr. C. Prakash has/day variation in fasting STC
However within normal range



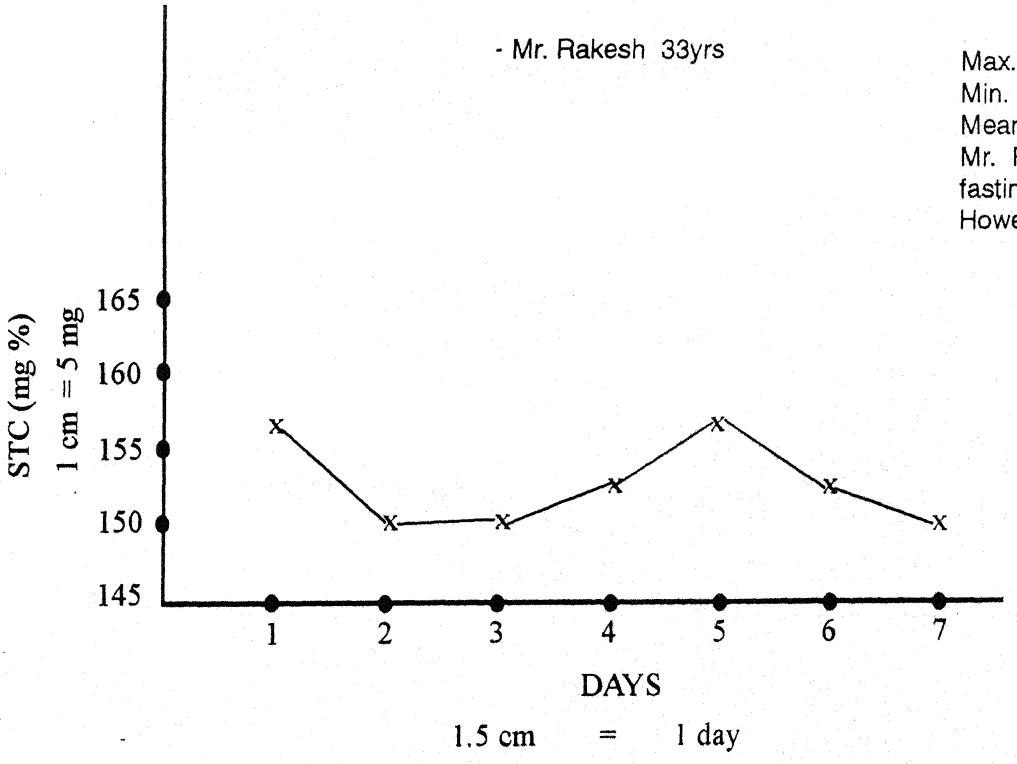
- Mr. Abad Lal 30yrs

Max. value = 172 mg
Min. value = 168 mg
Mean Value = 170± 1.52
Mr. Abad Lal has/day variation in fasting STC
However within normal range

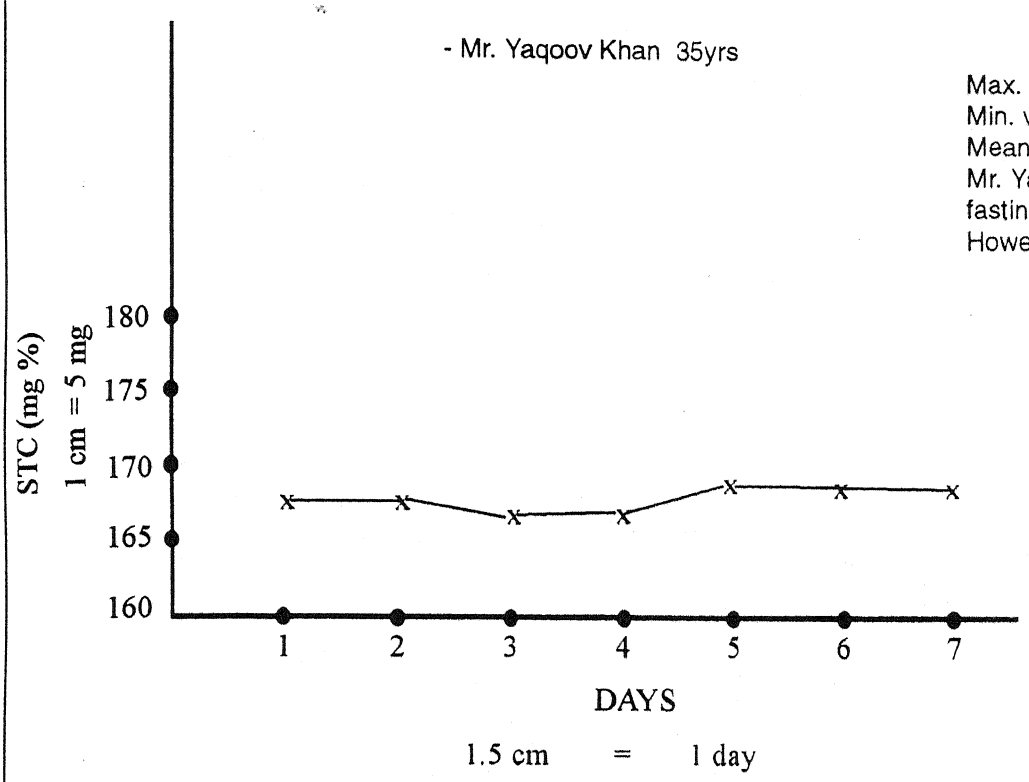


- Mr. Rakesh 33yrs

Max. value = 156 mg
Min. value = 150 mg
Mean Value = 152.28 ± 2.69
Mr. Rakesh has/day variation in fasting STC
However within normal range

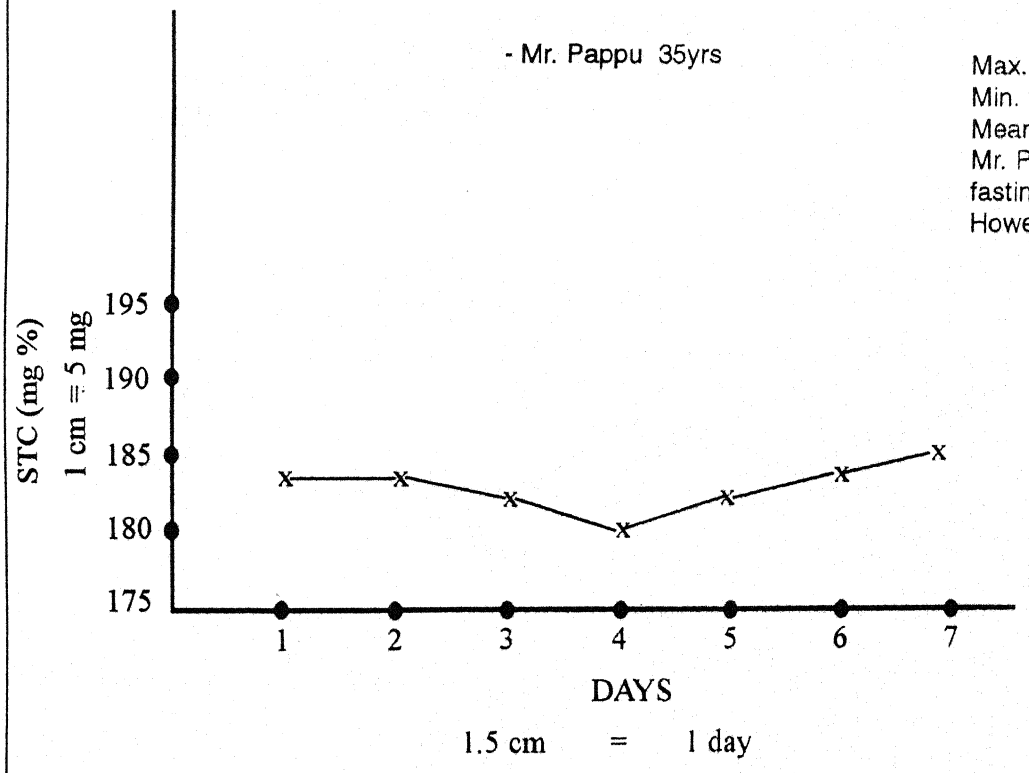


- Mr. Yaqoov Khan 35yrs



Max. value = 168 mg
Min. value = 166 mg
Mean Value = 167.14 ± 0.89
Mr. Yaqoov Khan has/day variation in fasting STC
However within normal range

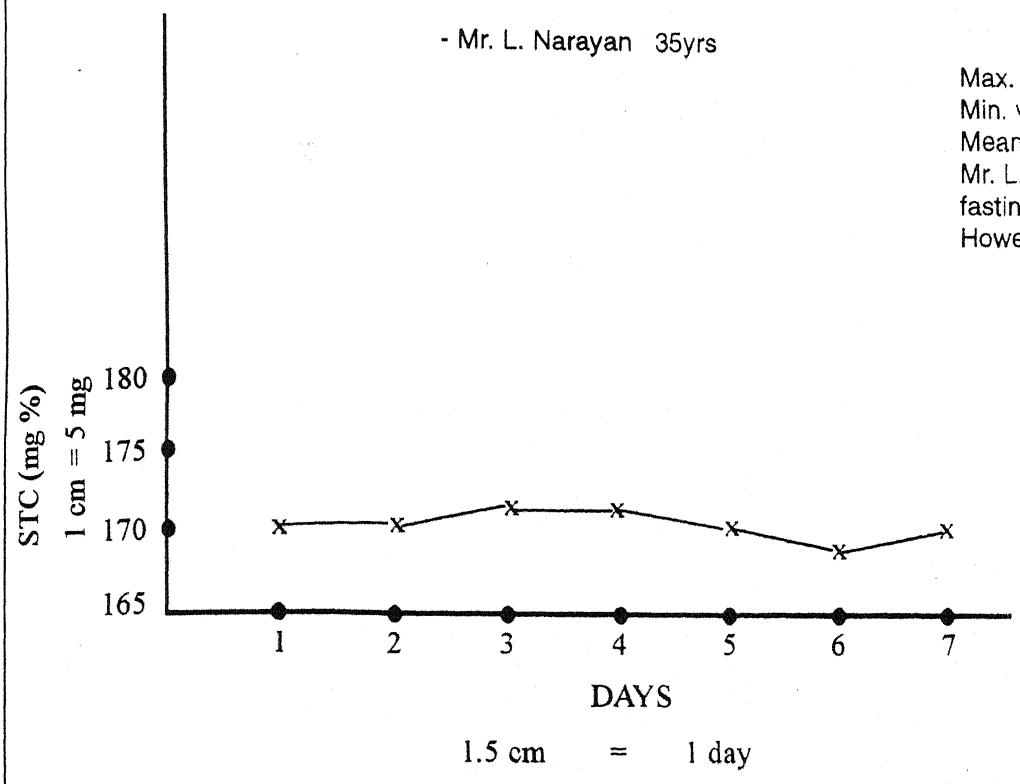
- Mr. Pappu 35yrs



Max. value = 185 mg
Min. value = 180 mg
Mean Value = 183 ± 1.73
Mr. Pappu has/day variation in fasting STC
However within normal range

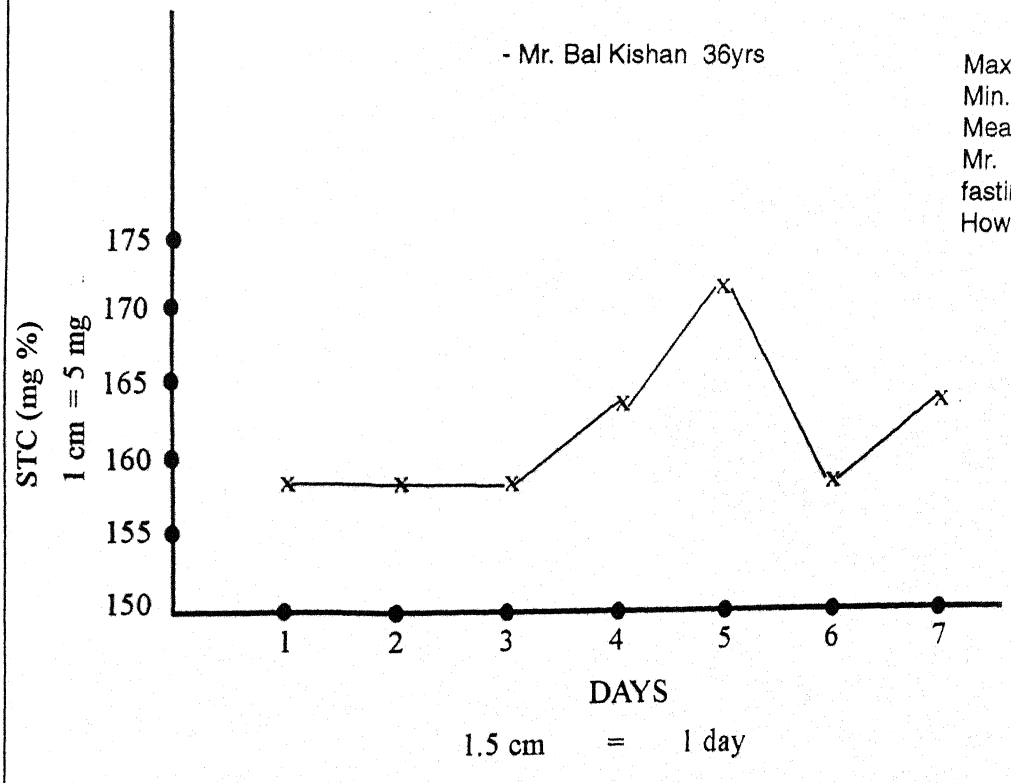
- Mr. L. Narayan 35yrs

Max. value = 171 mg
Min. value = 168 mg
Mean Value = 170± 1
Mr. L. Narayan has/day variation in fasting STC
However within normal range



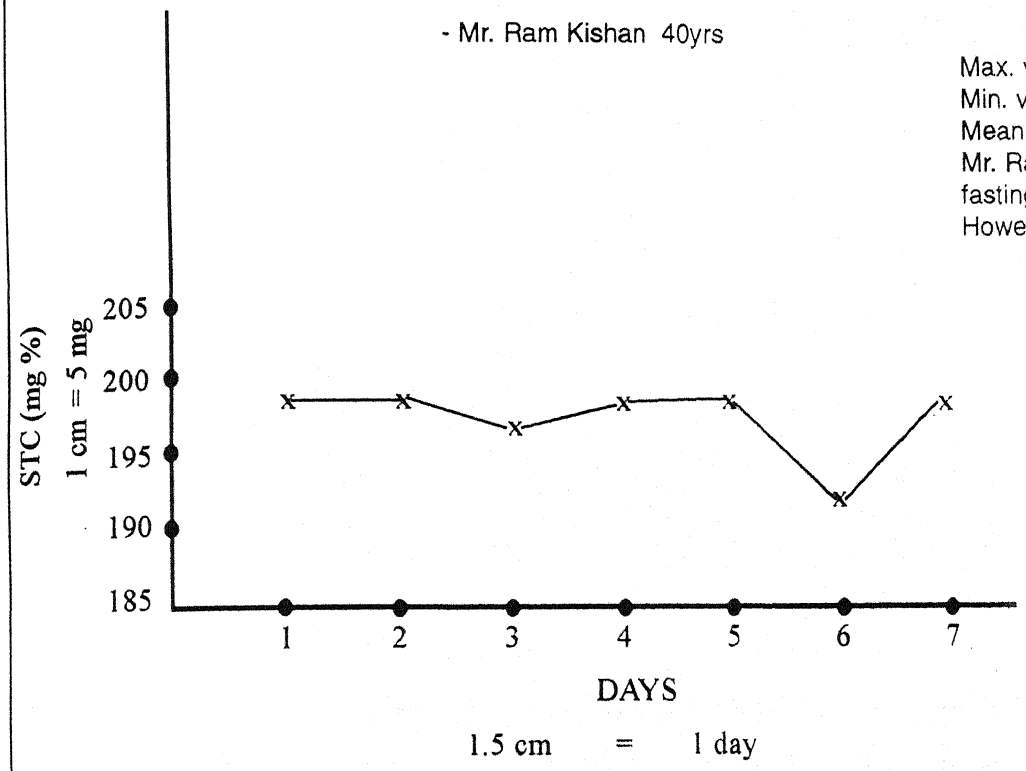
- Mr. Bal Kishan 36yrs

Max. value = 171 mg
Min. value = 157 mg
Mean Value = 161 ± 5.50
Mr. Rakesh has/day variation in fasting STC
However within normal range



- Mr. Ram Kishan 40yrs

Max. value = 198 mg
Min. value = 192 mg
Mean Value = 196.85 ± 2.26
Mr. Ram Kishan has/day variation in fasting STC
However within normal range



SUMMARY & CONCLUSION

Summary and Conclusion

The present study included 32 subjects none of them proved cases of Hypercholesterolemic on the basis of fasting STC.

All Subjects showed a large day by day variation in fasting total Serum cholesterol however, serum total cholesterol values were within normal range.

The female subjects age range (20-40 yrs) mean age 30.6±6.44 years showed a large variation in fasting serum total cholesterol as compared to male subjects age range (22-40 yrs) mean age 28.76±5.85 years.

Various group study showed a marked fluctuation in serum total cholesterol after single fat diet feeding or even treatment with anti hypercholesterolemic drugs.

Thus in our opinion we must be cautious about interpreting the group data shows benefits or harms of various dietary measures or drugs on lipoproteins because in this study we have observed that there are fluctuations in fasting serum total cholesterol when done even daily without treatment or any dietary changes.

So changes in fasting serum lipoproteins when done after weeks or months apart in open trails may themselves shows greater

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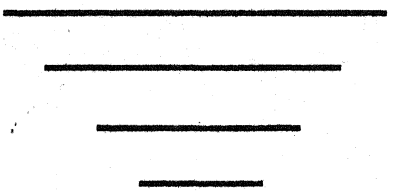
Thus in our opinion we must be cautious about interpreting the group data shows benefits or harms of various dietary measures or drugs on lipoproteins because in this study we have observed that there are fluctuations in fasting serum total cholesterol when done even daily without treatment or any dietary changes.

So changes in fasting serum lipoproteins when done after weeks or months apart in open trails may themselves shows greater

fluctuations, this should always be taken into consideration when accepting the benificial claims of drugs which show few % of rise of High Density lipoproteins (HDL) or fall of LDL by few % as proof of their efficacy.



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APPENDIX

WORKING PROFORMA

DEPARTMENT OF MEDICINE, M.L.B. MEDICAL COLLEGE, JHANSI

Dated :

NAME OF INVESTIGATOR	:	RAM SEWAK
NAME OF THE GUIDE	:	Asst. Prof. Navneet Agarwal, M.D.
NAME OF THE CO-GUIDE	:	Prof. R.C. Arora, M.D., D.Sc.
Place of Investigation	:	Hospital -
		Ward/Bad -
Date of Examination	:	

A. **PERSONAL HISTORY**

1. Patient's name :
2. Age/Sex :
3. Religion :
4. Address :
5. Weight :
6. Height :
7. Socio-economic status :
8. Sector : Rural/urban :

B. **RISK FACTORS**

a) Non-modified :

b) Modified :

i) **Major -**

1. Smoking
2. Tabacco chewing
3. Obesity
4. Alcoholism
5. Hypertension
6. Serum cholesterol/Serum triglycerides.

ii) **Minor :**

1. Emotional state
2. Fat diet
3. Diabetes mellitus
4. Family history of P/M, CAD.

C. CHIEF COMPLAINTS

1.
2.
3.

D. PRESENT ILLNESS

E. GENERAL EXAMINATION

General condition			Cyanosis
Pulse rate			Jaundice
B.P.	:	Supine	Oedema
		Standing	Dehydration

Respiration

Temperature

CARDIOVASCULAR EXAMINATION

a. Inspection :
   - i) Precordium
   - ii) Apex beat
   - iii) Other pulsations

b. Palpation :
   - i) Apex beat
   - ii) Thrill

iii) Other pulsation

c. Purcussion :

d. Auscultation :

i) Heart sounds :

S1

S2

S3/S4

ii) Murmur Systolic

Diastolic

RESPIRAORY SYSTEM

C.N.S.

ABDOMEN

LAB INVESTIGATIONS

TLC Hb % :

DLC

CPK - MB

SGOT

SGPT :

Total S. Cholesterol :

CONCLUSION ON (OF BASAL CHOLESTEROL VARIATION)

Day 1st

2nd

3nd

4th

5th

6th

7th

Signature

MASTER CHART

MALE SUBJECTS

S.NO.	NAME	AGE yrs	FASTING CHOLESTEROL VALUE (mg%) (Days) 1	2	3	4	5	6	7	MAXIMUM VALUE mg%	MINIMUM VALUE mg%	DIFF. mg%	MEAN ±SD mg%
1.	Mr. Ashok	22	180	180	184	184	188	185	180	188	180	8	183±3.10
2.	Mr. Ram Prasad	22	168	166	168	168	166	166	165	168	165	3	166.71±1.25
3.	Mr.Ram Kishan	22	152	152	154	152	150	152	150	154	150	4	151.71±1.38
4.	Mr. Vaheed	23	185	188	188	185	185	185	180	188	180	8	185.14±2.67
5.	Mr. A. Kr. Agnihotri	23	171	157	171	157	152	152	155	171	152	19	159.28±8.26
6.	Mr. Jagdish	25	177	177	182	185	182	170	178	185	170	15	178.71±4.88
7.	Mr. Rajesh Kumar	26	164	164	164	168	171	171	168	171	164	7	167.14±3.18
8.	Mr. Rohit	26	164	165	166	164	170	162	164	170	162	8	165±2.51
9.	Mr. Kalka Pd.	28	172	172	171	172	174	170	171	174	170	4	171.71±1.25
10.	Mr. C. Prakash	28	162	167	162	165	165	162	165	167	162	5	164±2
11.	Mr. Abad Lal	30	168	171	168	17	172	171	170	172	168	4	170±1.52
12.	Mr. Rakesh	33	156	150	150	152	156	152	150	156	150	6	152.28±2.69
13.	Mr. Yaqoov Khan	35	167	167	166	166	168	168	168	168	166	2	167.14±.89
14.	Mr. Pappu	35	184	184	182	180	182	184	185	185	180	5	183±1.73
15.	Mr. L. Narayan	35	170	170	171	171	170	168	170	171	168	3	170±1
16.	Mr. Bal Kishan	36	157	157	157	164	171	157	164	171	157	14	161±5.50
17.	Mr. Ram Kishan	40	198	198	196	198	198	192	198	198	192	6	196.85±2.26

Mean ± SD of Age of 17 Subjects 28.76 ± 5.85

Mean ± SD - of

Maximum Value	173.94 ± 11.48
Minimum Value	166.82 ± 1.50

MASTER CHART

FEMALE SUBJECTS

S.NO.	NAME	AGE yrs	FASTING CHOLESTEROL VALUES (mg%) Days 1	2	3	4	5	6	7	MAXIMUM VALUE mg%	MINIMUM VALUE mg%	DIFF. mg%	MEAN ±SD mg%
1.	Smt Bharti	20	171	171	178	178				178	171	7	174.5 ± 4.04
2.	Smt Suneeta	22	190	185	190	185	185	180	185	190	180	5	185.71±3.45
3.	Smt Vineeta	25	150	150	150	150	150	150	157	157	150	7	151± 2.64
4.	Smt Suneeta	26	178	175	182	180	180	178	178	182	175	7	178.71 ± 2.21
5.	Smt Kalpna	26	150	150	157	157	160	157	150	160	150	10	154.42 ± 4.27
6.	Smt kavita	28	150	150	150	150	157	157	150	157	150	7	152 ± 3.41
7.	Kr. Doly	28	185	185	185	177	180	185	185	185	177	8	183.14 ± 3.28
8.	Smt Babboraja	28	164	164	164	164	157	171	170	171	157	14	164.81 ± 4.63
9.	Smt Sudha	32	190	194	192	190	190	192	194	194	190	4	191.71 ± 1.79
10.	Smt Ram pyari	35	168	170	170	172	168	170	170	172	168	4	169.71 ± 1.38
11.	Smt Ram kumari	35	185	185	185	185	182	188	185	188	182	6	185 ± 1.73
12.	Smt Renu	36	167	171	170	171	170	170	170	171	167	4	169.85 ± 1.34
13.	Smt Phoola rani	38	155	155	157	157	152	152	155	157	152	5	154.71 ± 2.05
14.	Smt Susheela	40	164	166	166	168	168	164	166	168	164	4	166 ± 1.63
15.	Smt Kamla	40	200	185	185	192	192	185	200	200	185	15	191.28 ± 6.72

Mean ± SD of Age of 15 Subjects 30.6 ± 6.44

Mean ± SD - of

Maximum Value	175.33 ± 14.20
Minimum Value	168.2 ± 14.04